BRAIN.

JANUARY, 1882.

Original Articles.

ON THE POSITION OF THE MOTOR CENTRES IN THE BRAIN IN REGARD TO THE NUTRITIVE AND SOCIAL FUNCTIONS.

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In one pregnant sentence the German philosopher Goethe sums up the essence of human action. "Warum treibt das Volk so und schreit? Es will sich ernähren, Kinder zeugen, und sie ernähren wie es vermag." (Why are people so busy, and what are they roaring about? They want to feed themselves, to beget children, and feed them as best they can.) In these three reasons for human activity we find that one is personal and that two are social. The first of these is the personal one, the duty of the individual to himself. The second and third are the social ones, the duties of the individual to society. The clear insight of the philosopher led him rightly to put the duty of the individual to himself first. Under certain conditions we find that the social may overpower the personal instincts, even in the lower animals, and lead the mother to sacrifice her own life in trying to save her progeny. In certain exceptional instances we find the social instinct extending beyond the circle of immediate blood-relations, and inducing a man, as in the well-known story of Damon and Pythias, to sacrifice his life for that of his friends, or even inducing him, like Sir Philip Sydney, to refuse the water which

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was offered to quench his own burning thirst, and give it to an utter stranger simply because he believed the other to need it more. Such instances as these do occur; but the care with which they are recorded, and the admiration with which they are regarded, clearly show how exceptional is their occurrence. The instinct of self-preservation is one which underlies, and, except in the very rarest cases, is more prominent than the social instincts. It may sometimes be brought out only in the utmost need; but when the survivors of a shipwreck are struggling for the last particle of food on board a raft, or for the last drop of water in the midst of the Sahara, or for a breath of air in the Black Hole of Calcutta, then the instinct of the individual to preserve himself, whatever may be the cost to others, makes itself manifest. The instinct of self-preservation, indeed, must be the fundamental one, for otherwise the evolution of the races of animals now living would have been impossible. Animals not having this instinct of self-preservation would necessarily have perished in the struggle for existence, and those endowed with it would have succeeded. The fundamental acts of self-preservation are the respiration of air and the acquisition of food. Respiration is a simple act, and, as the air is everywhere around us, very simple rhythmical movements are sufficient to carry it on. These movements, too, require no further development during life, and the respiration of the newly-born child is as perfect as that of the full-grown man, as far, at least, as the aeration of the blood is concerned. No doubt the adult learns to modify his respiratory acts for such purposes as singing and speaking, and the nerve-centres by which these modifications are affected are capable of gradual development by careful training. They are no doubt to be sought in the cerebrum, but the nervous centre through which the mere aeration of the blood is effected co-ordinates very simple movements only, requires little or no further development, and is situated in the medulla oblongata and upper part of the spinal cord. The centres by which the primary acts of nutrition, deglutition, digestion, &c., are effected, are also situated in the spinal cord and in the sympathetic ganglia, for an anencephalous infant will suck, and a pigeon deprived of its brain will swallow the grains of corn

placed in its beak, but they are unable to seek their food. The centres by which the more complicated acts required in order to obtain food are, no doubt, situated in the brain. Hence the anencephalous infant, or the animal deprived of its brain, are unable to seek food for themselves, although they may swallow it when it is placed within their mouths. Modifications of respiration in producing voice, although exceedingly important for men and animals in their social relations, are comparatively unimportant for the nutrition of the individual, whereas the movements for seeking and obtaining food are essential to its existence. We should therefore expect that the motor centres in the brain would be arranged chiefly with reference to the acquisition of food, and that the centres would be modified in different animals according to the manner in which they obtained their food. That this is really the case appears, I think, from an examination of the functions of the motor centres of the brain as ascertained by Ferrier:

1. The eyes open widely, the pupils dilate, and head and eyes turn towards opposite side.

2. The opposite arm and hand extend forward as if to reach or touch something in front.

3. The opposite arm is retracted and adducted, the palm of the hand being directed backwards.

4. The fore-arm is supinated and flexed, so as to raise the hand to the mouth.

5. The angle of the mouth is retracted and elevated.

6. The ala of the nose and the upper lip are elevated, and the lower lip is depressed, so as to expose the canine teeth on the opposite side.

7, 8. The mouth is opened and the tongue protruded and retracted. The mouth is alternately opened and closed, and the tongue moves. The movements here are distinctly carried out bilaterally.

9. The opposite angle of the mouth is retracted, the platysma myoides is thrown into action, and when this is powerful the head is drawn slightly to the side.

(*a*), (*b*), (*c*), (*d*). Individual and combined movements of the fingers and wrist, ending in clenching of the fist. Centres for the extensors and flexors of the individual digits could not be

differentiated, but the prehensile movements of the opposite hand are evidently centralised here.

10. Advance of the opposite hind limbs, as in walking.

11. Complex movements of the thigh, leg, and foot, with adducted movements of the trunk.

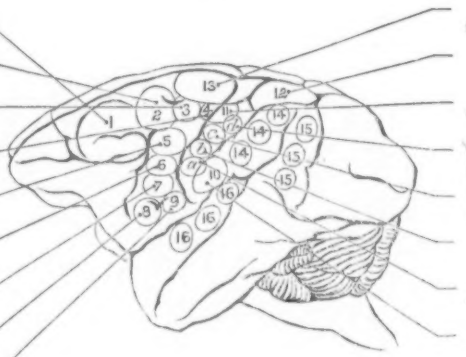
12. Movements of the tail, associated with some of the last movements.

If we look at the accompanying diagram, copied from Ferrier's work, with an alteration in the numbers by which he designates the different centres, we see that in the monkey the centres appear to lie around the fissure of Rolando in a definite order, corresponding to that which would be necessary for the animal to obtain food. Taking the centres in the order in which we have numbered them, we find that, when they are successively irritated, the sequence of movements is as follows:—that the eyes open widely, the pupils dilate, and the head turns as if to look for food. Next the hand and arm are extended as if to take it. The arm and hand are next retracted and adducted. Next, the hand is supinated and the fore-arm flexed, so as to bring the hand towards the mouth, as if to convey food into it. Next, the angle of the mouth is retracted and elevated, the canine teeth are exposed, the mouth is opened, the tongue retracted and protruded, and the mouth alternately opened and closed with movements of the tongue as during mastication. There is next retraction of the opposite angle of the mouth, with powerful contraction of the platysma myoides. I do not know whether this has any connection with the emptying of the cheek pouches in the monkey, but it is certainly an accessory act in mastication and deglutition. It is not observable in man under ordinary circumstances, but it at once becomes evident when deglutition is rendered difficult by sore throat and tonsillitis. A similar action is also noticed in man when powerful movements of the jaws are required, as in cracking a hard nut.

The next movements (*a*), (*b*), (*c*), (*d*), are complicated movements of the hand, which have not been more particularly analysed. Complicated movements of the hand are certainly required in frugivorous animals such as monkeys, in order to separate the edible parts of their food, as, for example, to pick out

FIG 1.

1. The eyes open widely, the pupils dilate, and head and eyes turn towards opposite side.
2. Extension forward of the opposite arm and hand, as if to reach something in front.
3. Movements of tail (and trunk).
4. Retraction with adduction of opposite arm.
5. Supination and flexion of the fore-arm, by which the arm is raised towards the mouth.
6. Action of zygomatics, by which the angle of mouth is retracted and elevated.
7. Elevation of ala of nose and upper lip.
8. Opening of mouth with protrusion of tongue.
9. Retraction of tongue.



13. Complex movements of thigh, leg and foot.
12. Advance of the opposite hind limb.
11. Retraction and adduction of opposite arm.
a, b, c, d. Prehensile movements.
10. Retraction of opposite angle of mouth.

BRAIN OF MONKEY.

the kernel from a nut which they have just cracked. The centres are absent in the dog, where no such movements are wanted.

The next movement is that of retraction and adduction, and is the same as that which has already been noticed as being employed in gathering food. I have numbered the centre for this movement 11, and it is the same as that which I have numbered 4. Ferrier has reckoned these two centres as one. I have, however, taken the liberty of dividing his single centre into two, inasmuch as it extends over the fissures of Rolando, and as it appears to me that this movement, although of the same character, is employed for a double purpose. It seems to me that while the movement of retraction and adduction started by centre No. 4 is for the purpose of gathering food, the same movement is related, when started by centre 11, to the casting away of the refuse portions of food. I have not had an opportunity of observing the habits of monkeys since I began to write this paper, but, so far as my recollection serves me, the movement of the monkey in throwing away refuse is somewhat peculiar, nut-shells being cast by these animals, not to the right side, as they probably would be by us, by an outward movement of the hand, but down to the left by a quick movement of retraction and adduction. Next, the leg is advanced as in walking, (10) and then come complex movements of the thigh, leg, foot, and trunk, with movements of the tail in monkeys. These we may regard as movements of balancing, or possibly of climbing, in order to reach a new supply of food after the first has been consumed.

We have now finished the round of movements necessary for the procuring and masticating of food, and the cycle of movement again commences, with No. 1. On comparing the arrangement of centres in the brain of the dog or jackal with those in the monkey, we are struck by a curious difference. While in the monkey the centres are arranged in the order just mentioned around the fissure of Rolando in unbroken sequence, we observe that in the dog and jackal the centres which are arranged round the crucial sulcus, corresponding to the fissure of Rolando, are few in number, and that the others are situated lower down, on a different convolution. The

four centres surrounding the crucial sulcus in the dog, in Fig. 2, are numbered, in Ferrier's experiments, 12, 5, 4, and 1 respectively, corresponding to the centres numbered, in Fig. 1, 1, 2, 4, and 12 respectively. These centres are those for the direction of the eyes, movement forward of the fore-leg, its retraction and adduction, and advance of the opposite hind limb. These are the movements required for seeing food, and for simple running straight forward in active pursuit of the fleeing animal which is to supply it. The use of the lateral movements of the tail in pursuit is seen in the grey-hound, where the tail aids the animal in guiding its rapid course or in turning; but as it also is not so essential as the movements of the limbs, nor in such constant action, its centre, No. 3, Fig. 2, is placed somewhat aside from the principal gyrus. We see here that the arrangement of the centres in the dog and jackal differs from that in the monkey very much in the way we would expect, for while the monkey, living in trees yielding abundance of fruit, has only to see where the food is hanging and by one or two efforts to reach it, animals of the dog tribe have usually, after seeing their prey, to run for a considerable distance before they can hunt it down, and bring into action the muscles of the mouth and jaws by which they seize and devour it. The centres for sight and for running are, then, arranged in simple order round the crucial sulcus, while those for the movements of the jaws are placed in an adjacent convolution (Fig. 2). In his 'Expression of the Emotions,' Mr. Darwin has noted the action of the muscles of expression in protecting the eye from injury or strain, and one of the points that strikes us most forcibly in the arrangement of the motor centres in the dog is the action of the zygomatics and orbiculares in causing closure of the eyes, this closure being very closely associated with movements of the mouth. I do not know whether wolves or jackals shut their eyes at the moment they fix their fangs in the flesh of their prey, but such a movement as this would seem to be well adapted for the protection of their eyes, either from injury by the animal they attack, or from the effects of the increased tension caused by the muscular efforts in the act of attacking. On looking at the brain of the cat we are struck

FIG. 2.

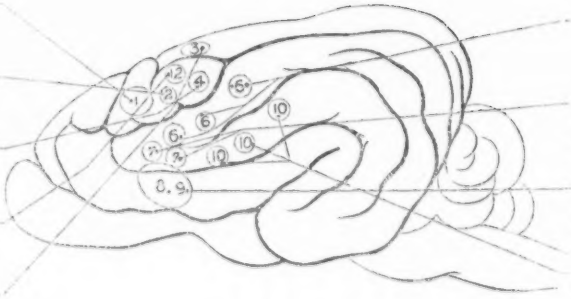
1. The eyes open, the pupils dilate, the eyes and head turn towards the opposite side.

2. The shoulder is elevated the opposite fore-limb extended forwards.

3. The opposite fore-limb is retracted.

4. The opposite hind leg is advanced as in walking.

5. Lateral movements of the tail.



6. Closure of eye.

7. Retraction and elevation of angle of mouth so as partially to open mouth (? snarling movements).

8, 9. The mouth is opened and tongue protruded and retracted (licking movement), sometimes growling.

10. Retraction of angle of mouth from platysma (? crushing bones or swallowing).

BRAIN OF DOG. (The numbers on the centres in the diagram correspond to those in Fig. 1.)

by the absence of the centre No. 12 in Ferrier's work, corresponding to No. 1 in the diagram in this paper, the centre which causes movement of the eyes. At first sight this seems very extraordinary, because the formation of the eyes of the cat is so peculiar that one is accustomed to think that it hunts its prey very much by sight. The want of this centre seems to me to render it probable that the sense of hearing and the tactile sense furnished by the cat's whiskers are considerably more important in directing the animal towards its food than even the sense of sight. We see, also, that the centre 4, for the retraction and adduction of the fore-arm, is much more developed than in the jackal or dog, and this we should conclude from the use which the cat makes of the fore-arm in striking its prey. On looking at the centres in rabbits, guinea-pigs, and rats, we see that they are arranged in the order 2, 4, 12, in this diagram, and 5, 4, 1 in Ferrier, that is to say, extension and retraction of the fore-leg, and forward movement of hind-leg. There is also great extension of the centres for the movements of the mouth, just as one would expect in rodents.

From this arrangement in these different animals it appears, I think, that the motor centres are arranged round the crucial sulcus or fissure of Rolando in such an order as, first of all, to subserve the wants of the animal in obtaining food, the motor centres which are first called into requisition in going after the food being nearest to the fissure, and those which are only required after the first centres have been in action for some time being arranged in an adjoining convolution. The social instincts, and especially the sexual, which is the primary social instinct, are subserved more especially by those modifications of respiration which give rise to voice. We find animals of the cat tribe purring or growling over their food, yet the voice is not of primary importance in obtaining food. Its chief use is that of enabling the animals to communicate with each other. Such communication is no doubt primarily of sexual importance, but it is also useful in enabling animals of the dog tribe, such as wolves, to obtain food by summoning one another to collect together for the purpose of hunting in packs. Although there are some very marked exceptions

amongst animals, it is, generally, only after the wants of the individual have been satisfied by food that the sexual instinct comes into play, and we should therefore expect that the motor centres connected directly or indirectly with it, such as the centres for voice, would be further removed from the crucial sulcus, or fissure of Rolando, than the motor centres required for the obtaining of food. This we find to be actually the case, but I am not at present in possession of sufficient data to enable me to extend my paper to this branch of the subject.

Even should the conclusions at which I have arrived, and which I have here stated, be erroneous, the hypothesis that the motor centres are arranged in the way I have described will enable those who have experienced difficulty in remembering their position to do so with ease, as I have had occasion to observe in the case of students.

For convenience of reference I give here in tabular form the corresponding centres as numbered in Ferrier's work on the Functions of the Brain, and in Figs. 1 and 2 in this paper.

Ferrier.	Figs. 1 and 2.
1	12
2	13
3	3
4	4
5	2
6	5
7	6
8	7
9	8
10	9
11	10
12	1

METHODS OF PREPARING, DEMONSTRATING, AND EXAMINING CEREBRAL STRUCTURE IN HEALTH AND DISEASE.

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(Continued from p. 360.)

Examination of Internal Structure of the Brain.

1. INTRODUCE a large section knife into the longitudinal fissure, and cut outwards across each hemisphere, about ¾ inch above the corpus callosum, exposing the *centrum ovale minus.* Repeat the same procedure on a level with the corpus callosum, exposing the greater centrum ovale, and exhibiting the continuation of the transverse strands of this great commissural tract with the medulla of the hemisphere. The student will now proceed to note the relative and absolute amount of grey and white matter, and inquire into the consistence, colour, and other physical qualities of these parts in the manner already detailed.

2. By an incision carried from before backwards across the fibres of the corpus callosum, a short distance on each side of the median line, the lateral ventricles are opened. To expose the structures more fully, divide the corpus callosum anteriorly and whilst gently reflecting it cut backwards, with a scissors through the vertical septum (septum lucidum), which is attached to its under surface, and which, descending, divides the lateral ventricles into two cavities. After reflecting the corpus callosum and dividing across behind, note the condition of the following structures seriatim—

a. The caudate nuclei or intra-ventricular portions of the corpora striata.

b. Septum lucidum, enclosing betwixt its walls the fifth ventricle.

c. Exposed anterior tubercle of the thalamus opticus.

d. Stria cornea, or exposed part of the tænia semicircularis coursing between the thalamus and corpus striatum.

e. Fringed margins of the velum interpositum or choroid plexus of lateral ventricles.

f. Fornix, and behind its descending pillars, the foramen of Monro.

g. General condition of lining membrane or ependyma.

3. Divide the fornix anteriorly close to its descending pillars, and after examining the subjacent velum interpositum, reflect both structures backwards, exposing the thalami optici, separated by the intervening third ventricle, and note the condition of the following parts:—

a. Grey matter forming the boundaries of the central cavities.

b. Anterior, middle, and posterior commissures.

c. Pineal gland and its ganglionic extensions, or peduncles.

d. Corpora quadrigemina (nates and testes).

e. Follow out the fornix posteriorly into the descending cornua of the ventricles.

4. On separating the occipital lobes from the cerebellum the curved bistoury may, by a little careful dissection, be made to cut through both *crura cerebri* obliquely upwards, to meet *above the corpora quadrigemina* at an obtuse angle in the third ventricle in front of the posterior commissure.

By so doing we separate from the cerebrum the cerebellum, pons, and medulla, together with the *corpora quadrigemina* and *pineal gland*. This method is certainly preferable to dividing the crus at the level of the superior cerebellar peduncle, which is so often done at the sacrifice of the normal relationships of most important regions.

5. Separate the cerebral hemispheres by median section through the slight remaining connections, and proceed thus:

a. Slice one of the hemispheres in a direction parallel to the sections already made when exposing the centrum ovale. Let numerous sections in this direc-

tion be made at different planes from above downwards through the whole hemisphere, so as to expose the structure of the thalamus and corpus striatum down to the base.

b. Slice the remaining hemisphere in a direction at right angles to its long axis, or, in other words, from above downwards, beginning on sections near the olfactory bulb, and proceeding as far back as the occipital lobe.

The sections (*a* and *b*) will familiarise the student with the *relationships and coarse structure* of the basal ganglia, points of considerable moment. Examine, therefore, by aid of these sections the medullary tracts forming the *internal and external capsules*, the *lenticular and caudate nucleus* of the corpus striatum, the *thalamus opticus* and its various regions and environment. All these parts may be studied in succession, referring to good illustrations, such as appear in the last edition of Quain's 'Anatomy.' The intimate structure and relationships of these regions will be dealt with further on.

6. Remove the cerebellum from its attachments by dividing the three peduncles, and proceed to examine successively

a. Superficial aspect of membranes covering pons and medulla.

b. Condition of blood-vessels and superficial origin of the cranial nerves.

c. Condition of valve of Vieussens, aqueduct and fourth ventricle.

d. By transverse sections expose structure of nates and testes, of the pons, its anterior, or motor and posterior or tegmental tract, of the medulla oblongata, noting the relative dimensions and appearance of the olivary bodies, the pyramidal and restiform tracts.

7. Lastly, after examining the lobules and membranes of the cerebellum, divide it into an upper and lower half by an incision carried through both hemispheres from behind forwards, exposing the central medulla.

Vertical section, i.e. at right angles to the lamina of the cerebellum, will display its foliated arrangement, and betwixt vermiform process and the middle of each hemisphere we pass through the plicated corpus dentatum.

Minute Examination of the Brain.

Prior to considering in detail the methods adopted for the minute examination of the brain, the student should be fully impressed with the conditions which render many of the processes applicable for other tissues quite inadmissible here. The consistence of cerebral tissue is such that cutting fine sections by the hand, either with an ordinary razor or the Valentin blades, cannot be adopted with any hope of success, since the finest sections thus obtainable are useless for microscopic examination, which requires sections of extreme tenuity. These extremely fine sections can only be obtained by aid of the screw microtome from portions of hardened or frozen brain. In order to modify the consistence of the tissue so as to adapt it for this purpose, various methods of hardening by chromic acid, chrome salts, alcohol, picric acid, and osmic acid have been devised. The results which have accrued from these methods have been most valuable, yet they are uniformly open to the serious objection that this altered consistence induced by the reagent is obtained at the expense of modified structure and altered relationships. Amongst the most serious drawbacks to the use of corrugating reagents in the examination of the brain are the following :—

A considerable expenditure of time (from three to eight weeks being required for satisfactory hardening).

The process itself is extremely tedious, and often unsatisfactory in its results, as evidenced by experienced histologists.

It requires a considerable amount of practised manipulation, and exposes the tyro to numerous disappointments and failures.

The shrinking of tissue is a most objectionable feature in the cortex, often amounting to one-half the original bulk.

The normal wealth of structure is greatly modified; in this respect it is infinitely surpassed by fresh methods.

The absolute and relative depth of the various layers of the cortex is subjected to most objectionable modifications.

It affects the cortex of different animals to a different extent, thus interfering with comparative investigations. Many pathological and normal appearances are wholly obliterated by the "hardening processes."

Of all tissues, that of the brain, from its extreme delicacy and susceptibility to rapid post-mortem change, is the tissue which, above all others, demands a rapid, ready and fresh method of preparation, the employment of indifferent media, and the restriction of all corrugating reagents. No method as yet adopted can surpass for elegance, expedition and certainty the freezing method; and, above all, it is *the* method which should be chiefly trusted to in all exact anatomical and pathological research. Yet, whilst we claim for the fresh freezing methods such self-evident advantages over the older processes, the long series of objections to the latter tabulated above must not induce us to blind our eyes to the fact that it also possesses its own advantages and cannot be dispensed with. Most of our classical descriptions of cerebral structure refer to brain which has been subjected to these methods of preparation; and few indeed, far too few, are the delineations of the minute structures of the cortex and medulla in the perfectly fresh brain. Hence, as a question of simple comparison between the results of different observers and our own, the hardening methods must still be adopted. There are numerous other very apparent reasons why we cannot afford to dispense with the older processes, and the student is therefore recommended, whilst guarding himself from its fallacies, checking or confirming its results by the fresh methods, to make himself equally familiar with the practical details of both methods, and to learn to discriminate the special worth to which each process is more particularly suited.

Two distinct methods of Freezing.—Just as we find amongst the list of reagents used for hardening brain some, and especially osmic acid, far less open to the objectionable qualities possessed by alcohol and chromic acid, inducing in fact very slight alteration in the relationships and bulk of the tissue, so do we find the different methods of freezing possess their own distinct and relative merit. This fact, which it is all important to recognise when dealing with structures like the

brain, has been wholly misunderstood or overlooked by all authorities who have written upon the subject, and we constantly find histologists grouping the freezing by ice and salt with the ether-freezing method, as though both were equally suitable for all tissues alike. Now the fact is that the ordinary *ice and salt* freezing-microtome is entirely useless for brain-structures, except after a modification of the latter by a process of hardening, and hence no longer a *fresh* method. Upon the other hand, the ether-freezing microtome is applicable to all tissues having a consistence not above that of the liver, but is pre-eminently adapted for nervous tissue. I find it necessary to insist upon this point, since it seems generally understood that the ice-and-salt mixture is suitable for freezing brain. Prior to the introduction of freezing by ether, practised histologists constantly complained of the impossibility of obtaining fine sections of brain by freezing, the objections being that hard spiculæ formed within its structure, and tore it up on cutting through it. Very lately this fact has been again asserted by a skilled manipulator,[1] who has even introduced a method of combined hardening and freezing to overcome this very unsatisfactory action. Hitherto, therefore, it may be asserted that the use of the ice and salt microtome has failed for the purposes of the cerebral histologist; and although by further development it may be rendered subservient to his purposes, for the present the freezing of brain substance must be uniformly pursued upon the ether-freezing microtome. The methods available for the examination of the minute structure of the brain are as follows:—

Hardening processes for sections to be cut on the imbedding microtome.

Hardening processes for sections to be cut on the freezing microtome.

Fresh process by means of the ether-freezing microtome.

Fresh process by a modified teazing and staining.

Fresh process by ordinary teazing with dissociating reagents.

We will preface our account of these methods by a brief outline of the more useful microtomes used.

[1] Vide Hamilton in the 'Jour. Anatomy and Phys.' vol. xii. page 259.

Microtomes for Imbedding.

Amongst the various instruments devised for cutting sections of hardened tissues are the microtomes of Henson, Rivet-Leiser, Brandt, Roy, His, Ranvier, Stirling and Rutherford. The instrument more generally used in this country is that devised by Stirling, and its modification by Rutherford, either of which instruments will prove satisfactory to the student; whilst that of Ranvier may be employed where it is desirable to grasp the microtome in the hand, or immerse it during section-cutting in water or spirit. The principle adopted in Stirling's instrument has been very generally followed. In the three last named, the body of the instrument consists of a metal tube or hollow cylinder, fixed to a table by some simple arrangement, or held in the hand in an upright or vertical position. A fine micrometer-screw works into the lower end, driving through the hollow of the cylinder a closely fitted piston-plug, which in its turn propels the mass in which the tissue to be cut is imbedded. The essential portion of this form of microtome, therefore, is the body, or cylinder, and the powerful micrometer-screw. The body, or cylinder, includes the following divisions:—the hollow, or 'Well' of the cylinder; its closely fitted plug, or 'Piston;' the upper smooth and levelled extremity, or 'Section-plate;' the lower extremity forming a "female screw" for the reception of the micrometer-screw. It is far preferable that the cylinder should be clamped by projecting arms and screw to a firm support as a table. In choosing a microtome for cutting sections of an imbedded tissue the student must be guided by the following considerations. The instrument should be strong but compact, and not weighty or cumbersome; it should possess appliances for fixing it firmly and immovably to a supporting ledge or table. Its well should be at least one inch in diameter, and the oval section is to be preferred to the circular well, since this form of well wholly prevents rotation of the imbedded mass. The section-plate should be absolutely level, perfectly smooth, polished, and show no irregularities of surface or indentations around the margin of the well, which otherwise would ruin the edge of the blade. The micrometer-

screw should be of powerful leverage, work evenly, easily, and without the slightest "loss of time," and have a pitch of at least fifty threads to the inch. Allowing his judgment to be guided by the above rules, the student cannot err greatly in his selection of a microtome for ordinary work and the smaller class of sections. A few words on the different varieties of instruments used may prove of service to the student.

Stirling's Microtome is a compact, strong, and admirably finished instrument, embracing all the qualities essential for the section-cutting of hardened preparations. For small sections no better adapted form has yet been devised, and for his earliest attempts in cutting hardened brain the student is recommended to secure this form of microtome. This instrument is made to be clamped to a table, so that both hands are free for section-cutting.

Ranvier's Microtome.—This is a smaller but very useful instrument. It has, however, to be held in the hand, a defect which is in part counterbalanced by the ease with which it can be immersed in spirit, and sections cut whilst so situated. It is well adapted for sections of spinal cord and the large nerve-trunks.

Rutherford's Microtome.—This, which is a modified form of Stirling's microtome, is a most valuable instrument, as it is well adapted for section-cutting of imbedded or of frozen tissues. It will be described fully in the section on freezing-microtomes. It has the disadvantage of being somewhat cumbersome as compared with the smaller "Stirling," whilst at the same time as a freezing instrument for nervous tissues it is excelled by the ether-freezing microtomes. The latter remark does not apply to other animal tissues.

Roy's Microtome[1] consists of an inner vertical brass plate covered with a layer of cork, and sliding by vertical movement within an outer brass-framework. A glass rod of horse-shoe form is fixed horizontally by its extremities into the brass frame, upon which the blade glides during section-cutting. The imbedding mixture is first cast in a zinc mould, the tissue being placed in it, and when hardened, the mass is fixed upon the cork plate. The vertical movement of the brass-

[1] Described and figured in the 'Journal of Physiology,' Vol. 2, No. 1.

slide is obtained by propulsion from a fine-threaded screw fixed below. A simple tubulature, adapted to the instrument, allows spirit or water to be blown upon the mass when required. It has been stated that tissues may be cut fresh with this microtome after freezing by the direct application of the ether spray. The student must be cautioned against any such attempt; for all tissues alike, the direct application of ether is to be deprecated, and for brain and spinal cord and all nervous structures such a method is wholly inadmissible. Roy's microtome has the disadvantage of not being readily fixed to a table during manipulation.[1]

Schiefferdecker's Microtome is described here as applicable to hardened preparations, although it is essentially constituted to dispense with the method of imbedding. The object to be cut is fixed within a hollow brass cylinder by means of a clamp upon its surface worked by two screws. In lieu of a propelling screw below, as in other microtomes, a circular plate forming the section plate can be elevated or lowered by the revolution of an outer concentric plate which turns upon a screw. The student will find this microtome figured and described in the 'Quarterly Journal of Microscopical Science' for January, 1877.

Microtome for slicing through whole hemisphere of Human Brain.—The instrument which I have made for this purpose at the West Riding Asylum consists of a heavy brass cylinder, three inches deep and eight inches in diameter, closed in below, where, however, its cavity communicates centrally with that of a small secondary cylinder—the screw-socket. In this socket, which is four inches deep, works a powerful and finely threaded screw, having a milled head three inches in diameter. A flat, circular brass plate, accurately fitted to the interior of the upper cylinder or well, is raised or lowered by means of the screw, the movement being communicated to the imbedding mass, which rests upon it above. The section plate is contuted by the projecting rim of the cylinder above, and is mathematically level and smoothly polished. The microtome rests upon the iron collar of a powerful tripod stand. A zinc

[1] The instrument is made by Mr. Gardner, South Bridge, Edinburgh.

tray, two and a quarter inches deep and twenty-six by seventeen inches, can be adapted to the microtome-cylinder, so that sections may be cut under water with the greatest ease.

Microtomes for Freezing.

These, as before stated, are either ether, or ice-and-salt freezing instruments.

The Ether-Freezing Microtome (Bevan Lewis).—This microtome, which I believe is pre-eminently suited for the purpose of the cerebral histologist, was described in the pages of the 'Journal of Anatomy and Physiology' for April 1877, and as modified and improved twelve months later, in the October number of 'BRAIN,' to which I must refer my reader for a detailed account of the instrument and the method of freezing. The section plate is either made of metal, or consists of plate-glass drilled with a central aperture through which the freezing chamber glides. I would strongly recommend the instrument which has the plate-glass top, as it forms a great safeguard against injuries to which the section-blade is subject from scratches and indentations on the metal surface. A further improvement has been introduced by the maker, in the form of a lateral support for the ether-spray apparatus, communicating with a bellows worked by the foot. In the latter form the spray adjustment is easy, and the hands are both free for section-cutting and manipulation, and thus a most substantial and valuable improvement has been obtained with but a very trifling addition to the cost of the microtome.[1]

Rutherford's Freezing Microtome.[2]—This instrument consists of a brass section-plate, with a central aperture leading into the interior of a vertical tube, in which a plug is fitted to move upwards or downwards by means of a fine screw. The cylinder is surrounded on all sides by a metal box covered with gutta-percha, which holds the freezing mixture of ice and

[1] These improved microtomes may be obtained of Mr. Gardner, 45, South Bridge, Edinburgh, who, at a minimum of charge, has fully perfected the workmanship.

[2] "A New Freezing Microtome." 'Monthly Micros. Journal,' vol. x. page 185. 'Outlines of Practical Histology.' By William Rutherford, M.D., 2nd edit. page 164.

salt. An exit tube allows of the escape of water. The whole instrument can be securely clamped to a table. The method of using this instrument for freezing is as follows:—The plug is first unscrewed and oiled, so as to prevent its fixture during the process of freezing. The tissue to be frozen and cut is first immersed in a thick solution of gum for some hours, "in order that the gum may permeate every part of the tissue, and prevent the formation of a crystalline condition within the frozen tissue." If the tissue has been previously immersed in alcohol, all traces of the latter should be first removed by soaking in water. Equal parts of *finely powdered* ice and salt are now placed in the freezing-box and stirred around the well, whilst the latter is filled with the solution of gum. As the gum freezes around the periphery, the tissue is plunged into it, and held until it is fixed by the advancing ice. Care should be taken to keep the exit tube of the box open, so as to allow of free escape of water, and to close the freezing box by a weighted strip of cork, to prevent the entrance of heat and to exclude the salt of the freezing mixture. By this method the freezing process may be accomplished in from ten to twenty minutes. All delicate tissues require a *special preparation* to adapt them for this process, otherwise the water which they contain freezes into hard icy spicules, which tear the structure when it is cut. Dr. Pritchard has therefore recommended a prior immersion of the tissue in thick solution of gum-arabic, using the same solution as the imbedding material.

Quite recently, Dr. Hamilton writes as follows with regard to freezing the nervous structures, and especially brain, by means of the ice-and-salt microtome. "It was found that the crystals of ice so broke up the delicate nervous tissue as to render it totally useless for minute examination. . . . I attempted two years ago to modify the method of freezing, but without success, and accordingly gave it up as an almost hopeless task."[1]

According to this statement, the writer's experience tallies wholly with my own; and fully acquainted as he was with the methods of steeping tissues in gum prior to freezing which were adopted by Dr. Rutherford, it is clear that this method

[1] 'Journal of Anat. and Phys.' vol. xii. page 257.

of freezing was not applicable for fresh brain, although it answers admirably in the case of firmer and less delicate textures. Dr. Hamilton overcame the difficulty of freezing brain with the ice-and-salt mixture, but at the expense of sacrificing the fresh for the chrome-hardening processes, a special procedure being requisite, which will be considered further on. Now it is after such considerations that we recognise the undoubted superiority of the ether-freezing microtome for nervous structures. It has been stated that the ether-freezing process has been entirely superseded by the introduction of ice and salt, or other mixtures, which by *constant* refrigeration keep the mass in a frozen state for hours. All practical manipulators, however, know that this is, as regards nervous structures, a fallacy and a blunder, since the very excellence of the ether-freezing method depends upon the fact that it can be checked at any stage and renewed when required. This is the all-important consideration in freezing brain, for beyond certain limits it is frozen into a hard icy solid, which at once blunts or turns the edge of the blade; but with the ether-spray this stage need never be attained, and a consistence is obtained admirably adapted for section-cutting. The student should therefore bear in mind that *the constant application of a freezing mixture to fresh brain cannot but result in failure for section-cutting;* and that *by use of the ether process the prior preparation of brain by hardening, or by immersion and saturation in mucilaginous fluids, may be wholly dispensed with.*

Williams' Freezing Microtome.[1]—In this instrument a wooden tub containing a freezing mixture of ice and salt is covered by a glass lid, the frame of which is secured by a clamp screw. An upright brass conducting-bar passes from the interior of this box through a central aperture in the lid, and to the extremity of this bar is screwed the circular brass plate which supports the tissue to be frozen. The arrangement of the knife is peculiar, since it is fixed in a triangular frame, and can be either raised or lowered by the screws which are adapted to the frame. There are three additional plates for

[1] This instrument is made by Messrs. Swift & Sons, University Street, London.

supporting the frozen object, and a brass cup for holding substances, which are fixed in cacao-butter or paraffin.

The box is filled with equal parts of pulverised ice and salt, care being taken to prevent the mixture touching and so fixing the cover. After the cover is replaced and screwed down, the substance to be cut is placed on the central brass plate, surrounded by a little solution of gum, and the apparatus is covered with baize to facilitate freezing. When frozen, raise the blade, and after the first cut across, proceed as follows:—Each end of the razor must be presented to the surface of the section, and *exactly levelled by means of each of the back screws.* If the large back screw be now turned, the blade can be lowered to any required extent; and since a complete revolution of the screw gives us a section $\frac{1}{100}$ of an inch in thickness, and the screw-head is graduated into sixths —a movement through sixty degrees gives us a section $\frac{1}{600}$ of an inch thick.

The special features of this microtome, in which it differs materially from others already described, consist in an arrangement whereby the blade and not the imbedded or frozen tissue becomes the movable part of the instrument; and the edge of the knife is only brought into contact with the substance to be cut.

Preparation of Hardened Brain.—In subjecting the brain to the agency of hardening reagents certain important conditions should always be kept in mind, as they are essential to success. The conditions are as follows:—

(*a*) The reagent should act equably upon all portions of the tissue.

(*b*) The requisite consistence must be acquired at the expense of the minimum of alteration and shrinking of tissue.

Now the first of these conditions can only be obtained by ensuring a thorough saturation of the tissue throughout, so that the fluid permeates rapidly to the central or deepest portions of the mass. It is evident that the surface of the tissue being bathed in the reagent will be more actively affected by the latter than the more distant parts within, and thus arises the danger of a too rapid hardening of the exterior, which forms a mechanical impediment to the permeation of the deeper

structures by the surrounding fluid. Always, therefore, take the precaution to ensure a *free and rapid permeation of the tissues by the fluid.*

Again, if any portion of the surface is in close contact with the side of the containing vessel, it necessarily is less affected than the surface bathed in the fluid. The mass should therefore be so suspended that it is *equably bathed all around in the reagent.*

Another important consideration is that of temperature, for under the most favourable circumstances the central or deeper structures must remain far less subject to the action of the reagent than the exterior of the mass, and are, therefore, the parts which most readily succumb to putrefactive changes. The larger the mass, therefore, to be hardened, the more difficult is it to prevent central decay, whilst an elevated temperature induces the same result. The tissue to be hardened should therefore be of *moderate bulk* as compared with the fluid in which it is immersed, and the preparation should be kept in a cool spot, or, better still, *in an ice safe.*

The next condition we have named, and which it is equally important to secure, is that a minimum of shrinking of tissue should result from the action of the reagent, and it is a well-known fact that all the reagents used for this purpose will, if employed in too concentrated a form, ruin the preparation by inducing extreme shrinking and brittleness of the mass; whilst again, some of these reagents are far more reliable than others, and less open to these disadvantages. The more commonly used hardening reagents are—osmic acid, Müller's fluid, solutions of the chrome salts, chromic acid, picric acid, methylated spirits, and alcohol. Now this list represents their relative value as hardening reagents for nervous tissues, osmic acid and Müller's fluid standing at the head of the series as the most valuable and least injurious in their action; chromic acid and alcohol occupying a far less prominent position in the scale. In order therefore to secure the tissue from injurious shrinking, employ in preference to the others the *reagents noted at the head of the list,* and use the *weakest solutions compatible with safety to the tissue,* commencing with the weakest, and gradually augmenting the strength of the solu-

tion, or later on, even replacing by the more astringent reagents. Let us now detail throughout the process recommended to the student for his first essay in chrome-hardening, subsequent to which it will be useful to briefly dwell upon the various modifications of the process adopted by others.

Process of Hardening by Chrome. (*a*) *Müller's Fluid and Potassium Bichromate.*—Excise a portion of the ascending frontal or parietal convolution of human brain, cutting across its length so as to remove about an inch of the convolution along with its central and deeper medulla. Lightly cover it all round with a little cotton-wool, and immerse it in from two to three ounces of methylated spirits contained in a four-ounce stoppered bottle.

Label the bottle with the name of the specimen and date, placing it in a cool cellar or ice safe.

In twenty-four hours pour away the spirit, replacing it by four ounces of Müller's fluid, the preparation being surrounded as before by cotton-wool to ensure it being bathed upon all sides alike by the fluid.

Let the bottle stand in a cool spot, and in three days replace the fluid by a fresh quantity. At the end of one week the fluid should be again renewed, or, preferably, a weak solution of potassium bichromate substituted (2 per cent.) At the end of the second week a solution of the latter of double the strength may be added; and if at the termination of the third week the mass is still pliable, and of the consistence of ordinary rubber, it is as yet unfit for section-cutting, and the reagent should be replaced by a solution of chromic acid.

In these later stages the chromic acid expedites the process without producing the extreme shrinking of tissue which ensues if it be used at earlier stages. For the same reason even absolute alcohol may at this stage be employed, but although valuable in the preparation of the spinal cord, it cannot be equally well recommended for the cortex of the hemispheres. By the above process our preparation will have attained the requisite consistence within a period of from four to eight weeks. It will be observed that in the various steps of this process the conditions previously emphasised as essential to success are obtained. Thus the early immersion in methylated spirit

abstracts all superfluous fluid from the brain and its vessels, and entering its substance by its affinity for water, aids in the rapid permeation of the mass by the chromic solution in the next stage. The more prolonged action of the spirit would, however, prove highly detrimental. Again, the cotton-wool ensures an equable distribution of fluid around, whilst the comparative bulk of the mass and fluid in which it is immersed, and the temperature to which it is exposed, provide for equable hardening to its deepest structures, and ensure it against decomposition. The gradual increase in the potency of our reagents from Müller's fluid to the strong solutions of chrome are also adjuncts to the hardening of the central portions, whilst we also expedite the process of hardening.

(*b*) *Potassium Bichromate and Chromic Acid* (Rutherford).—Place small portions of the cerebrum in methylated spirit for twenty-four hours, observing the same precautions as to relative bulk of the preparation and the reagent, covering with cotton-wool, and leaving in a cool place. Replace the spirit by a mixture of potassium bichromate and chromic acid. The proportions may advantageously be varied, according to the condition of the structure to be hardened; but the solution recommended by Rutherford, and which answers well, contains 1 gramme of chromic acid, 2 grammes of potassium bichromate to 1200 C.C. of water. Change at the end of eighteen hours, and then once a week. Should the tissue not be sufficiently tough for cutting at the end of six weeks, place it in a $\frac{1}{6}$ per cent. solution of chromic acid for a fortnight, and then in rectified spirit.

(*c*) *Iodised Spirit and Potassium Bichromate* (Betz).—Large portions of cerebrum may also be placed for a few hours in methylated spirit, tinted of a light sherry-brown by tincture of iodine. Add fresh iodine solution as the colour fades. In one or two days remove the pia mater, and return the preparation to the solution, adding to the latter half its bulk of fresh iodised spirit. After the lapse of another period of two days, replace the solution by iodised alcohol (alcohol 70°–80°) tinted of a sherry-brown by tincture of iodine). In from two to three days it should be transferred to a 4 per cent. solution of potassium bichro-

mate until sufficiently hardened for section-cutting. Should a brown deposit form over its surface during the process, let it be well washed, and a fresh solution used. When hardened, these preparations may be kept permanently in ·5 per cent. solution of bichromate. If the cerebellum is to be hardened, the segments are at once placed in the iodised alcohol, adding fresh iodine frequently as the colour of the solution pales. Remove the pia mater on the second or third day, and in a week transfer to pure methylated spirit for twenty-four hours, and finally harden it in a ·5 per cent. solution of potassium bichromate. This process, recommended by Prof. Betz, is not suitable for examination of the cortex, as the iodised spirit is injurious to the after-processes of staining. It is, however, especially suited for obtaining large sections through the hemisphere, the whole cerebrum and cerebellum, when sliced evenly across into segments ¾ of an inch thick, being most satisfactorily hardened throughout. For the minute examination of the cortex methods (*a*) and (*b*) are pre-eminently to be preferred.

(*d*) *Müller's Fluid and Ammonium Bichromate* (Hamilton).—This method is especially applicable to large segments of the brain, and is much to be preferred to the process by chromic acid (*e*). The brain is sliced completely through into segments about one inch thick. Each segment may now be placed in a large vessel, such as a brain-preparation jar, padded with cotton-wool, and containing a comparatively large bulk of the solution, which consists of three parts of Müller's fluid to one part of methylated spirit. A refrigerator or ice safe should invariably be employed to preclude decomposition, and the pieces should be turned over occasionally in the solution. In about three weeks' time they may be transferred to a solution of ammonium bichromate (·25 per cent.). At the end of the fourth week replace by a 1 per cent. solution of the same salt, and the following week by a 2 per cent. solution, in which they remain until fit for section-cutting.

(*e.*) *Chromic Acid Solution* (Lockhart Clarke).—The convolutions of the cerebrum and cerebellum were by this process hardened in a ·25 per cent. solution of the crystallised chromic acid—a stronger solution rendering them far too brittle for

section-cutting. As stated above, chromic acid is not adapted for hardening so satisfactorily and uniformly as the chrome salts. This method, employed by Lockhart Clarke, has been superseded by more reliable methods, as are also the processes recommended by Stilling, Kolliker, Hanover and Van-der-Kolk, for the hardening of nervous textures.

Hardening by Osmic Acid (Exner).[1]—It has been already stated that osmic acid (OsO_4) is one of the most reliable agents for hardening the brain and other nervous textures which we possess. Prof. Sigm. Exner of Vienna has therefore devised a method whereby *small* portions of the brain may be prepared for section-cutting by means of this reagent.

A small portion of brain, not exceeding one cubic centimetre in size, is placed in ten times its volume of a solution of osmic acid (1 per cent.). The solution should be replaced by fresh reagent of the same strength after the lapse of two days—a proceeding which may be advantageously repeated at the end of the fourth day. In from five to ten days the piece is usually stained throughout, for this reagent has the valuable property of hardening and staining simultaneously. The hardened brain is then washed in water, plunged for a second in alcohol to facilitate the imbedding, and sections are cut in the usual way in an ordinary microtome. The subsequent treatment of these sections will be given later on, when methods of staining and mounting are considered. It is only necessary here to remind the student that osmic acid is really a most valuable agent in the investigation of brain-structure, and he should avail himself of every opportunity of becoming familiar with its action.

Summary of Processes for Hardening.—The student having been thus placed in possession of the more valuable methods for preparing the brain for the imbedding microtome, it will be well to indicate the method he should adopt in his earlier attempts, as each process has its own individual merits. It is advisable that his first attempts should be made with comparatively small portions of tissue—about 3 C.C. in bulk—and that he should employ the process of hardening by Müller's fluid and potassium bichromate.

[1] Zur Kenntniss vom feineren Baue der Grosshirnrinde. Aus dem lxxxiii. Bande der Sitzb. der k. Akad. der Wissensch. iii. Abth. 1881.

(*a.*) This process, to which I assign the first place amongst the chrome-hardening processes, is slow but very certain. The results are, to my mind, more satisfactory than those of any of the other methods, and I invariably adopt it myself as the one for general use at the West Riding Asylum. The vessels which the student will find most convenient to use for hardening these smaller portions of tissue are the stoppered-glass bottles of four-ounce capacity used for dispensing purposes. Each bottle should be labelled with the name of specimen and date and nature of each successive change of reagent, whilst the specimen should occasionally be removed, and its consistence noted so as to familiarise the touch with the increasing firmness of the tissue, and the degree of hardness requisite. The method recommended by Rutherford (*b*) is also very reliable for moderate-size specimens. The methods advocated by Betz and Hamilton (*c*, *d*) are peculiarly well adapted for large segments of the brain, a whole hemisphere being thus readily hardened throughout. When these large masses of tissue have to be dealt with, special precautions are requisite. Thus the hemisphere should be sliced horizontally or vertically into segments not over $\frac{3}{4}$ of an inch in thickness. Each segment should rest upon a bed of cotton-wool in the vessel for its reception; and if two segments are included, another stratum of cotton wool should be interposed betwixt them. The vessels containing the specimen should be of large size, varying in capacity with the bulk of the latter. Thus a large preparation glass, such as is used for preserving brain in spirit on museum-shelves, will be found well adapted for hardening the whole hemisphere; whilst pickle-bottles, especially those provided with the "patent lever stopper," are most suitable for the basal ganglia, cerebellum, smaller portions of the cerebrum and the pons. Still smaller segments, such as the student will have more frequently to deal with, are best hardened in the four-ounce stoppered bottle, a good supply of which should be kept on hand. All the foregoing processes are contrasted with that of Exner's (osmic acid) in being most adapted for the demonstration of the nerve-cells and plexuses of the grey matter of the brain and its ganglia; whilst the latter, failing in this respect, is, on the other hand,

infinitely better suited for exhibiting the structure of the cerebral medulla and its extensions into the cortex of the brain. The student is therefore recommended to make himself acquainted with the structures of the cortex by the chrome methods, and subsequently to employ Exner's method for demonstrating the medullated tracts of the cortex.

Imbedding and Section-Cutting. — Having satisfied himself that the specimen is sufficiently hard for section-cutting, the student first cuts off a portion which can be accommodated by the well of the microtome, and prepares it for imbedding by a prior immersion for a few minutes in rectified spirits. This removes the fluid derived from the chrome solution from which it has been taken. In the meantime the imbedding mass should be melted at the lowest temperature requisite. The microtome-screw should be lowered sufficiently to allow of a deep imbedding of the tissue. The preparation is next removed from the spirit, rapidly dried by a fold of blotting-paper, and momentarily plunged into the warm imbedding mixture which upon its removal leaves a film over its surface, whilst all small cavities or lacuna which it may contain are filled up. The melted mass is now poured into the well of the microtome, and the preparation immersed and held in the position required for section-cutting until fixed by the cooled and consolidated mass. If the imbedding mass is one subject to much contraction on cooling, it is requisite, just as it is becoming "set," to press down with the end of a spatula the margin of the wax and oil mass against the sides of the well.

Imbedding Mixtures. — In the process of imbedding, the principle to be remembered is that the mass should be not too resistant to the blade, whilst at the same time it affords efficient support for the imbedded tissue. If the texture be one readily permeated by the imbedding mixture, contraction of the latter in cooling is apt to result in injury to the preparation, more especially if this be one of the more delicate structures. Thus in the delicate embryos of the fowl and other similar organisms, special methods of imbedding will be requisite. For the brain and spinal cord, however, the process is comparatively simple, since

the imbedding mass neither permeates its structures nor injures it by contracting.

The following are the more important media employed for imbedding prior to section-cutting:

1. White wax. Olive oil, equal parts.
2. White wax, 3 parts. Olive oil, 1 part.
3. White wax. Cacao-butter.
4. Solid paraffin, 5 parts. Hog's lard, 1 part.
5. Solid paraffin, 5 parts. Paraffin oil, 1 part. Hog's lard, 1 part.
6. Solid paraffin, 5 parts. Spermaceti, 2 parts. Hog's lard, 1 part.
7. Spermaceti, 4 parts. Cacao-butter, 1 part.
8. Spermaceti, 4 parts. Castor oil, 1 part.

The first on this list is perhaps the more generally employed medium, but it possesses the disadvantage of contracting strongly on cooling, so as to leave an interval betwixt the wax plug and the sides of the well: this, of course, will necessarily tend to loosen the mass, and allow rotatory or wobbling movements, which prevent accurate section-cutting. These disadvantages may be overcome by one or other of the following measures:

Vary the proportion of the oil and wax in favour of the former. When solidifying, press down the edge of the mass with a spatula. Use a microtome plug with groove or projections on upper surface. Drive the mass out of the well, partly surround it with a narrow strip of blotting-paper and return it—forcibly pressing it down into the well—the blotting-paper swells by imbibition of the spirit in section-cutting, and so fixes the plug firmly.

In the use of the third medium, viz. white wax and cacao-butter, far less contraction ensues upon solidification, and a firm supporting mass is obtained, the proportion of the ingredients varying with the firmness required. I can also speak favourably of the combination of paraffin with hog's lard. In all these cases it is well to keep the mass in a small tinned pot with lip, provided with cover and handle, so that it can readily be held over a gas-jet or Bunsen burner, and melted as required. Another imbedding agent used is gum, which is solidified by the application of alcohol or

methylated spirits. This method, proposed by Brücke, although valuable for many tissues, cannot be recommended for brain.[1]

Imbedding is wholly dispensed with in the form of microtome devised by Schiefferdecker, where the preparation having been hardened by alcohol as far as practicable, is placed in the well and clamped firmly by the arrangement already described. In the small hand-microtome of Ranvier, again, the ordinary melted media for imbedding are often exchanged for elder-pith, which is packed around the tissue in the well in a dry state, and then, upon immersion in spirit, the pith swells and firmly fixes the preparation. None of these latter methods are so suitable for hardened brain as imbedding in the wax and paraffin mixtures.

Section-Cutting.—The preparation being satisfactorily imbedded, and the mass perfectly cold and hard, our next procedure is to cut the *finest possible sections—they cannot be cut too fine.* Place on the table supporting the microtome, and in front of the latter, two vessels, one a Griffin beaker-glass of three inches diameter, and the other a cylindrical glass jar, a brain-preparation jar, or better still, a flat-bottomed porcelain evaporating basin. Nearly fill the beaker with methylated spirit, and the latter with water. Seat yourself in front of the microtome at a convenient level for the free play of the hands, place a soft towel over the knees, to be used for wiping off adherent wax from the imbedding mass. Have close at hand, also, a razor-strop, a porcelain capsule of small size, and some camel-hair brushes.

Dip the section-blade in the methylated spirit, and having raised the imbedded mass slightly above the level of the section-plate, slice off the superficial portion, exposing a clean smooth surface of the tissue for cutting. Again dip the blade into the spirit, and turning the microtome-screw, say through one-eighth of a revolution, place the flat of the blade upon the section-plate in front of the mass, with the back towards yourself and the handle in your right hand, grasped near the heel of the blade. Now cut your first section by a clean steady sweep from heel to point, and always *away* from your-

[1] 'Handbook of the Physiological Laboratory,' page 92.

self. No saw-like movement should be performed, nor should the blade be arrested occasionally in its course, the section shaved continuously and evenly off the preparation. The thin film of wax removed from the front of the tissue is sometimes likely to get in the way of very fine sections, and hence it is usually advisable, with a penknife to gently remove all the imbedding mass *in front* of the preparation to about the depth of a couple of millemetres prior to section-cutting. Subsequent sections should be cut with a still more restricted movement of the screw, until the operator has found the minimum degree of movement at which a section can be cut. This, of course, is a matter of tact, and the student will find that he daily improves the quality of his sections by practical manipulation. Two precautions must ever be taken if successful sections are desired. The first is, that as the blade is raised out of the spirit, the latter should not be drained off, but a good flow be retained over the surface of the blade, so that the section is floated up as it is cut. In the next place make it a rule to pass the blade across the strop frequently, after cutting half-a-dozen sections, as it requires to be kept at the finest possible edge. As the sections are cut they are floated off into the porcelain basin of water, and are freed from adherent particles of the imbedding mass by the rapid gyratory movements they here undergo from the currents caused by the attraction of water for the spirit they contain. After floating off each section in the water, pass the blade softly over the towel to clear its surface from adherent wax, prior to bathing it afresh in the spirit. How is the student to ascertain whether his sections be sufficiently thin? The exact thickness of the section is ascertained if the pitch of the microtome screw is known, and the degree of movement which he adopts noted. Thus, if the screw have eighty threads to the inch, a movement through one-eighth of a complete turn, i.e. 45°, will give a section $\frac{1}{640}$th of an inch in thickness, whilst a movement through an arc of 30°, or $\frac{1}{12}$th the entire turn, gives a section $\frac{1}{960}$th of an inch thick. He will, however, be not far from wrong by following this rule: reject all sections which do not float, and of the latter preserve as valuable only those which appear as a mere film on the surface

of the fluid, requiring to be looked at sideways to be distinctly seen. It is at this stage of the proceeding, that of section-cutting, that we can best judge of the merits of the imbedding mass, which, if found unnecessarily resistant to the blade, should be modified by the addition of more of the diluting adjuncts, viz., lard, oil, or cacao-butter.

The Section Blade.—This is an item of supreme importance to the histologist, and it is by no means true, as we often find asserted, that any razor blade answers the purpose equally well with the specially prepared knives. I have found this statement made frequently by those who were by no means fortunate in the thinness and delicacy of their sections; whilst all whom I am acquainted with, who can rejoice in the good qualities of their sections, have always paid great attention to the section-blade which they employ. The qualities of a good blade for section-cutting upon the microtome depend upon its make, form, edge, and tempering. It should be sufficiently long to ensure a free sweep over the whole surface of the section plate from heel to point, wide enough from back to edge to support and float up the largest-sized section which might have to be cut; it should be hollowed out on both surfaces, but be most concave on the surface uppermost in section-cutting; the edge should be extremely sharp and perfect, whilst the angle formed by the converging surfaces here should not be so acute as to involve bending of the blade when subjected to slight pressure; the quality and tempering of the steel should be of the best character. The above points are all most essential requirements in a good section-blade; but another important consideration is that the plane of the back and edge of the knife should exactly coincide when it rests upon a perfectly level surface as the microtome section-plate—not the slightest degree of tilting being admissible.

For the smaller microtomes I have invariably used razor-blades, my three instruments measuring in length and width of cutting-surface as follows:—

No. 1. 4 Inches × 1 Inch. (made by Weedon).
4½ „ × 1 „ („ by Young).
5 „ × 1½ „ („ by Young).

The first is a most valuable knife. It is fitted into a short,

firm, and fixed handle. The latter, which is my favourite blade, has a folding handle, and is sketched in outline with a section of its concave surfaces in an early number of 'BRAIN.'[1] Some of the earliest and most discouraging failures upon the part of the tyro in section-cutting depend upon the condition of the blade, and it is necessary that he should be fully impressed with the fact that not only must he secure a couple of section-knives with the above qualities, but that he should jealously keep them in most perfect order. To ensure the latter condition, let him observe the following precautions:—

Use no undue pressure against the section-plate.

Keep the surface of the blade absolutely level with the plate. Pass it frequently along the strop whilst cutting sections. After each day's work examine its edge most critically, and if the slightest irregularity or notching be apparent it must be reset before use.

Keep the blade dry and well polished by a soft handkerchief, and if in a closed handle, have a chamois-leather case made for it.

Subsequent treatment of Sections.—The sections have now to be removed carefully by a camel-hair brush to methylated spirit contained in the small porcelain capsule, in which they may be allowed to soak for some time to remove all traces of chrome, which otherwise interferes with the subsequent staining they have to undergo.

Large Sections through Hemisphere.—The above account refers especially to the smaller class of sections. When it is requisite to obtain sections upon a much larger scale, such as those through the hemisphere or the whole brain, the process requires modification. The method of imbedding is precisely similar in all its details, but when we come to cutting the section, special forms of knife are found requisite. The blade which I use measures 16 inches long by 2 inches wide, and is fixed in upright handles at each end. The zinc basin in which the large microtome rests is filled with water, and then sections are cut with a saw-like movement of the blade, and not, as in the former cases, by one complete sweep through the structures. It requires a steady hand and much manipulative skill to

[1] Vide 'BRAIN,' Part III. page 353. October, 1878.

obtain these larger sections both thin and perfect, and the student will only perfect himself by frequent and persevering efforts. These large sections can be readily removed by means of sheets of paper upon which they are floated, and this support may be given to them throughout the various subsequent stages of preparation. When the desired number of sections have been obtained, the imbedding plug may be removed, the remaining portion of tissue placed in methylated spirit, or a 5 per cent. solution of bichromate of potash, until required. The microtome should be carefully dried and placed away in a drawer for safety, as the slightest scratching of the polished section-plate will ruin a good blade in a few minutes.

The next stage through which our sections pass is that of staining and mounting, and this we had best leave for consideration in our next article.

ON THE BLENDING OF COLOURS BY THE SOLE AGENCY OF THE SENSORIUM.

BY JOHN GORHAM, M.R.C.S., TUNBRIDGE.

In order to obtain single vision with the two eyes, the images are known to impinge on identical or corresponding parts of the two retinæ; if these parts do not coincide, there is double vision. But the two eyes may be made to perform separate functions; the one to receive a colour-sensation, the other to discharge it.

There has been a good deal of controversy as to whether a true blending or mixture of two colours actually does take place in the brain.

The subjoined experiments show that it is possible to obtain a conduction or propagation of white or coloured sensation from one eye to the other, so that if a red light is presented to the right eye and a green to the left, the two sensations shall be united in the brain and call up that of yellow.

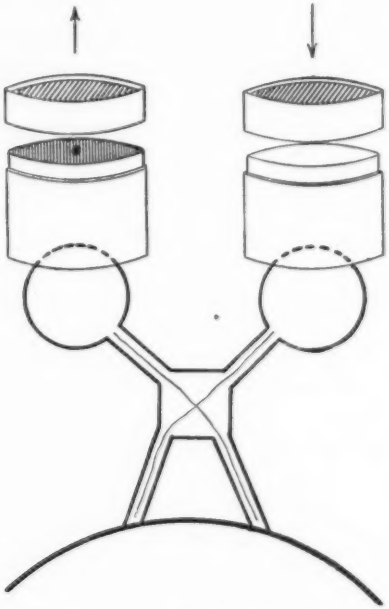
Such combinations I have found to take place when a large quantity of white or coloured light is caused to impinge on the retina of one eye, while a very small quantity falls on that of the opposite eye.

The only apparatus required is a couple of ounce boxes, which are converted into tubes by removing their lids and pushing their bottoms out; and four pairs of lids.

The tube used for the right eye is left open at both ends (diagram), and coated inside with lamp-black; that for the left eye is closed at one end by a thin metallic disc, perforated at its centre with a small needle the one-fiftieth of an inch in diameter. It is then blackened inside. The lids, having their tops removed, are mounted with coloured gelatine films, four

films to each lid; this forms a fair substitute for coloured glass, and is easily procured. Eight lids are used, and they are mounted in pairs, with green, blue, red, and yellow gelatine.

To simplify description, we will assume that the open tube is always applied to the right eye, the punctured one to the left.



EXPLANATION OF THE DIAGRAM.

The diagram shows the two lids mounted with discs of coloured gelatine, and the tubes through which light is admitted to the eyes. That for the right eye (to the right hand) is open at both ends, to admit much light; the tube for the left eye is closed at one end, and punctured with a fine needle, to admit of little light. Below are shown the two eyes engaged in looking through their respective tubes, with their optic nerves approximated in a square space (optic commissure) and having cross fibres which carry nervous energy from the eyes to opposite portions of the brain. The thick curve line at the bottom denotes a portion of the brain, from which these nerves derive their origin.

By this peculiar arrangement, light which is admitted to the right eye is found by experiment to take a circuit through the optic nerves and brain, finding its way to the opposite eye, and displaying itself in the disc made by the needle. This accomplished, the problem of *binocular* mixture is solved.

By this arrangement we impress both eyes with white or coloured light, the same in *purity*, similar or dissimilar in *hue*, but totally different in *degree*. The right eye is saturated with much light through a large orifice, the left with little light through a minute needle-hole.

The object of using tubes is to ensure that each eye shall be impressed with its own light unmixed with that which enters the other.

We first present a colour to the right eye, and white light to the left.

Closing the right tube by a lid of green gelatine, and admitting white light to the left eye through the needle-hole, we direct our gaze on a sheet of white paper spread open on a table.

Each eye has now become impressed with an image of its own; that seen by the right eye is a large round *green* disc, that by the left eye a small round *pink* one.

What is the cause of the pink colour of this little disc? that it is connected in some way with the sensation engaging the opposite eye there can be no doubt, for on shutting that eye the pink appearance of the little disc has vanished and is replaced by white light. On re-opening the right eye the white colour of the little disc is exchanged for pink, and this alternation of the two colours takes place as often as we choose to repeat the trial.

But our experiment is only half-completed; for when the two eyes are fully impressed with their colour respectively, if we now suddenly remove the green from the right eye the left disc loses its pink hue and becomes green.

Here, then, we have conclusive evidence from direct experiment that, when one eye is impressed with a colour-sensation, the other eye responds and participates in the effect; and, moreover, that this has not resulted from any external source, but from direct communication from eye to eye within the cranium through the brain itself. Corroborating evidence is obtained by trials with other colours.

We now cover the tube of the right eye with a lid of *blue*. Directing our gaze on a white surface, as before, we see a large *blue* disc, a smaller *yellow* one. On suddenly removing the

blue tube, keeping the right eye still open, the small yellow disc has as suddenly become blue.

Again: the right eye was similarly impressed with a *yellow* colour; its complementary *blue* was quickly imparted to the smaller disc seen by the left eye. But the simplest experiment of all is perhaps quite as conclusive as any.

We apply the tube to the left eye, and for a yellow film we substitute the yellow light emitted from a gas flame. Gazing on a white surface, with the right eye shut, the small disc is seen nearly white. We now open the right eye, when the small disc immediately assumes a beautiful cobalt-blue appearance, evidently the complementary of the light impinging on the opposite retina.

We arrive at the conclusion, therefore, that *when one retina is fully impressed with the sensation of a given colour, and the other retina feebly illuminated with white light, a current of colour is set up between the two eyes, and that this current is propagated from that eye which receives the most light towards that which receives the least. And, moreover, that which is transmitted to the left eye is first the complementary, and then the colour itself, of that which impinges on the right retina.*

On Sensorial Mixture when a different Colour is presented to the Right and Left Eye.—We place a lid of *green* film before the puncture in the left tube, and a lid of *red* film before the right eye.

Bearing in mind our former experiments, and utilising them in this, we are able to effect a combination of these two colours, just as when they are rotated on a wheel and form a yellow light.

Holding both tubes close to the eyes, and looking, as before, at a sheet of white paper, we observe first that the green of the small disc is intensified. This we anticipated; it has received the complement evoked from the red—we see, so to speak, green plus green. We now suddenly remove the red film from the right eye; the little green disc has changed to *yellow*. This, again, was confidently expected, for the original colour (*red*) being suddenly withdrawn from the right eye, it is then, and then only, that it appears to the opposite eye. It is at this juncture, therefore, that the two sensations really blend and form the binary *yellow* hue.

We do not always succeed upon a merely first trial, for the right eye often requires replenishing by a frequent application and quick withdrawal of the red tube. By this process of "feeding" the one eye, as Bacon has it, the little disc of the other gradually participates, loses its green appearance, and emerges finally into a pure yellow.

And if we reverse the position of the colours, placing the green before the right eye and the red before the left, the two blend as before, and form a brilliant yellow.

The yellow thus procured is as pure and well-mixed as that which is formed in one eye by the rotation on a wheel of equal sections of red and green. It contrasts strikingly with that kind of binocular mixture which has been attempted by the aid of the stereoscope, but with signal failure; the colours so nearly blending without actually doing so, that the appearance is most tantalising, while the individuality of each is never entirely lost.

To combine "Red" and "Blue."—We use a lid of *blue* gelatine for the left eye, and one of *red* for the right eye. Gazing on a white surface, we now observe a large red disc and a smaller blue one. This little blue disc is slightly deteriorated by receiving a green complementary from the opposite side.

We now suddenly remove the right tube and examine the effect this has produced on the blue disc. On repeating this process, say a dozen times, the blue has gradually become charged with red, and passes through a whole series of violet hues, from blue-violet to red-violet.

We have seen that in order to convert this blue disc into one of violet it must be made to combine with red, and also that this red sensation must, in some way, be procured from the right eye. Still the mode by which this is accomplished is remarkable, for it can be effected only by actually depriving the right eye at brief intervals with the very colour which impresses it, and by no other means. Hence, on withdrawing the red tube, its colour directly finds its way across to tinge the blue, and, by repeating the process, the blue gradually becomes charged with sufficient red to assume a violet hue. In this way we "feed," so to speak, the left eye with red, by

repeatedly withdrawing this colour which actually supplies it from the right eye.

To combine "Blue" and "Yellow."—These colours are complementary, and, when blended, form white light.

We apply a tube, armed with *blue*, to the left eye, and a *yellow* film to the right eye. Directing our gaze on a white surface, we see a large yellow disc, a small blue one.

The blue appearance of the little disc is seen to be enhanced by receiving the complementary of yellow from the opposite eye.

We now remove the yellow tube from the right eye. The effect of this is directly perceived; a propagation of yellow light takes place from right to left, and after a few repetitions the blue disc is completely merged in white.

To multiply experiments would be irksome to my readers, and encroaching on the pages of this Journal. It may be interesting to remark, in conclusion, that the complementaries, one and all, may be caused to blend, forming white light by impressing each eye with the *same* colour, when that of the little disc immediately merges into white. This result is inevitable indeed, and tends to confirm the legitimacy of the plan now proposed for showing that a true blending or mixture of two colours actually does take place in the brain, and that the mixture itself is always located, and may be perceived in a little disc prepared expressly for the purpose.

By the same arrangement as that shown in diagram, *motor* force, as well as *colour*-sensation, is found to be communicable from eye to eye, causing contraction and dilatation of the pupil. An account of this is reserved for a future communication.

A CLINICAL LECTURE ON SHAKING PALSY.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and Epileptic.

THE disease "Shaking Palsy," or "Paralysis agitans," some examples of which I shall have the opportunity of showing you, was first regularly described by our countryman Parkinson in 1817. Parkinson was a member of the Royal College of Surgeons, and his 'Essay on the Shaking Palsy' presents so graphic and admirable a description of the disease, that comparatively little has been left for subsequent observers to add to his account. In our time Charcot has also made the disease the subject of clinical investigation. The influence of these writers cannot fail to be felt at every turn by any one desirous of demonstrating the features of this remarkable affection. Parkinson's definition of Paralysis agitans is as follows:

"Involuntary tremulous motion, with lessened muscular power, in parts not in action and even when supported; with a propensity to bend the trunk forwards and to pass from a walking to a running pace: the senses and intellect being uninjured."

He quotes the distinction between kinds of tremor which was drawn by Sylvius de la Boe, who contrasts tremors produced by attempts at voluntary motion with those which occur whilst the body is at rest. Sauvages in 1763 had distinguished the latter species (Tremor coactus) by observing that "the tremulous parts leap and as it were vibrate even when supported;" whilst every other tremor, he observes, ceases when the voluntary exertion for moving the limb stops, or the part is supported, but returns when we will the limb to move.

We still depend very much upon this point—the difference of circumstances in which the tremor shows itself, for our diagnosis of Paralysis agitans.

The female patient, Mrs. G., is a widow, æt. 62. She sits with her chin resting on her chest, the lower lip and the neighbouring muscular structures about the chin being constantly in tremulous movement. There is a frown upon her brows. Her hands lie in her lap, the fingers being somewhat interlaced, apparently for the comfort of mutual support. When separated, the hands chiefly, and the forearms to a less extent, are in a state of constant tremor. If we count the to-and-fro movements, we find them to number from 160–170 in the minute. The tremor seems to be about equal in each hand.

When I speak to her and she answers, the shaking of the hands becomes more marked. It is not that the rapidity, however, is increased, but the movements occupy a larger space. My hand placed upon her knees cannot distinguish any trembling of the legs.

She can stand and walk, as you see, without help, but with short, "toddling" steps, the head being carried low and the body stooping forward. The face shows little or no expression, and wears a kind of fixed look. This attitude and general bearing it is very important to note carefully; they are characteristic of the disease. As she stands with her back towards me, if I pull her by the dress, with even the slightest force, she tends to fall backwards. When asked to lay hold of a cup which is offered to her, she puts out her right hand to take it, and whilst doing so the movements entirely cease. When, however, she has got hold of it and is told to squeeze the handle the movements return.

In another form of shaking palsy, that dependent upon insular, or multilocular, or disseminated sclerosis, as it is variously called, "the tremor," as Charcot has pointed out, "only manifests itself on the occasion of intentional movements of some extent; it ceases to exist when the muscles are abandoned to complete repose." The cessation of tremor in this woman when she stretches out her hand to take the cup is not an exceptional circumstance. In many cases of Paralysis agitans, it is true,

the tremor will persist throughout the voluntary movement. But the present feature also is common enough, and has not escaped the observation of Parkinson, who contrasts it with what happens in that which he styles "tremor." "It is necessary to bear in mind," he writes, "that this affection" (shaking palsy) "is distinguishable from tremor by the agitation in the former occurring whilst the affected part is supported and unemployed, *and being even checked by the adoption of voluntary motion;* whilst in the latter the tremor is induced immediately on bringing the parts into action. Thus an artist afflicted with the malady here treated of, whilst his hand and arm are palpitating strongly, will seize his pencil, and the motions will be suspended, allowing him to use it for a short period; but in tremor, if the hand be quite free from the affection, should the pen or pencil be taken up the trembling immediately commences."

Charcot, also, when referring to cases of shaking palsy in which the tremor only occurs intermittently, says: "Singularly enough, it is just in such cases that the tremor shows itself rather when the limbs are at rest, and ceases when they are set in motion by the will."

There can be no doubt that in general the persistence of the movements of the hands when the muscles are abandoned to repose, is sufficient to differentiate Paralysis agitans from insular sclerosis. This is a rule, however, which is by no means without exceptions. In one of my cases, which an autopsy showed was an example of insular sclerosis, the tremor occurred as well in a condition of repose as on the occasion of intentional movement; on the other hand, I think I can call to mind more than one case belonging probably to the class of Paralysis agitans in which the tremor was disposed to cease during muscular repose, and evince itself during voluntary movement of the limb.

The attitude of this patient's hands is another very noteworthy feature, and I would ask you to remember it particularly in reference to an examination we shall presently make of the other patient. It is, as Charcot has shown, very much the attitude of the writing hand, the fingers being inclined to an angle of about 45° with the metacarpal portion of the

hand. You will note, however, that the fingers do not present the easy curve seen in the writing hand. Their position, it seems to me, is precisely that which is obtained by faradising the dorsal interossii—the first phalanx is flexed, the second and third being extended upon the first. The tremulous movements of the thumb laterally, as well as backwards and forwards, are marked more strongly than those of the fingers, and convey the idea of the patient rolling some object between it and the fingers. The fingers, *en masse*, tend to deviate towards the ulnar side. The extended position of the fingers would seem to indicate a tendency to muscular rigidity as well as convulsion, and this is indeed what we find when we come to examine other parts of the body. It is especially well seen in the fixed position of the head and neck; the chin can be lifted, but it soon resumes its position. We also learn from the patient that she has a great tendency to become cramped in her limbs. This rigidity, as we shall see in the other patient, may become the most dominant feature of the case. When we take an aggregation of symptoms—*Symptomen-complex*, I think the Germans call it—and give a name to the disease which is characterised by the association of these symptoms, we must take care to remember that in various examples individual symptoms will certainly vary in their prominence. It will often happen that a symptom which perhaps we have thought of but lightly—have "thrown in" among the rest, as it were—will in a certain case assert itself so strongly as to dwarf those others, upon the presence of which we are accustomed to rely for making a diagnosis. On the other hand, a symptom which we have come to look upon as essential, may be actually absent. In this woman the presence of the peculiar tremor of the hands must be allowed to be, as indeed happens in the majority of cases of Paralysis agitans, the most striking feature. I have shown you, also, the slight indication there is of a tendency to rigidity of the muscular system. In her case this is a symptom pronounced only in the most trifling degree. We can readily imagine, however, that this symptom might be the most dominant one in a certain case, and that the tremor in its turn might be absent or only slightly marked. I shall be able to show you presently a man

who, I think, exhibits this kind of anomaly. But for the present let us return to the consideration of the disease of which we have a typical example in the person of this female.

More often than not, I think, the muscles of the face fail to show, to a great extent at least, the tremor which is so marked in the hands, but in this patient there is very distinct tremulous movement of the lips, especially the under lip. And in consequence of this inability to keep the mouth sufficiently closed, the saliva is almost constantly dribbling, much as we see it in cases of glosso-labio-pharyngeal paralysis. There is no difficulty, according to her account, in swallowing.

Mrs. G. suffers much from flushes of heat, and her face is ever and anon covered with a deep blush. In many cases these symptoms are accompanied by profuse general perspiration, but this is not observed in the present instance. An "habitual sensation of excessive heat" was described, for the first time, by Charcot as characterising Paralysis agitans.

There is a symptom in this patient which, according to my experience, is a very frequent one, but which has not hitherto, so far as I know, received attention at the hands of any observer. Charcot, it is true, has described the affection of speech, which is often observed, in these words: "There is no real difficulty of speech, but the utterance is slow, jerky, and short of phrase; the pronunciation of each word appears to cost a considerable effort of the will. If the tremor of the body be intense, it may happen that the utterance may be tremulous, broken, jolted out, as it were, like that of an inexperienced rider on horseback when the animal is trotting. However, in both cases we should recognise in this a phenomenon of transmitted tremor. Finally, the patients seem to speak between their teeth." We observe something of the kind in this patient, but the symptom upon which I now wish to speak concerns not the articulation but the voice. The voice has a peculiar "piping" character. It is, if you will observe, the conventional voice of the old man upon the stage. We all know that on the stage peculiarities of every kind are obliged to be accentuated for the sake of strong light and shade. When a very advanced period of old age has to be represented, the tone of voice adopted is exactly of the shrill

piping character which we note in this woman, who, however, is only sixty-two years of age. Since I first noted this "piping" note in cases of shaking palsy many years ago, I have been interested in observing whether it is common in the exceptionally aged persons (not afflicted with Paralysis agitans) whom from time to time I have met. I have not found it to be so. Now, Paralysis agitans, although, as Charcot has pointed out, it especially assails persons who have passed their fortieth or fiftieth year, has a slow march and little or no tendency to curtail longevity. Hence there are always about a certain number of persons afflicted with Paralysis agitans who have arrived at a ripe old age, and who present this peculiarity of voice. I cannot help thinking that the conventional voice of age on the stage has originally been derived from the study of some old person affected with this disease, and thence handed down, as we know is the stage custom, to successive generations of performers.

A slight tremulousness shows itself in the patient's tongue. There is no affection of the cutaneous sensibility, nor of the special senses. The bowels are very obstinate, so that she is habitually obliged to take aperient medicine. She can retain the fæces, but has to be very quick in relieving her bladder, and requires to get up five or six times every night for the purpose. She suffers pain of an aching character in her limbs.

We do not observe in this patient a symptom which is so common in this disease as often to enable us to recognise a case at a glance. Parkinson describes it as "a propensity to bend the trunk forwards and to pass from a walking to a running pace." Although the patient presents the fixed head and stooping body, she does not hurry along as we so often see in such cases. There is no "festination," to use the term employed to designate this hurried march.

The amount of muscular power varies much in individual instances, and also in different stages of the disease. At first there may be but little notable weakness; but as time goes on, a very distinct loss of power shows itself in the limbs and especially in those affected by tremor. In this woman the power of grasp is only feeble.

Before proceeding to consider other points connected with the symptoms of this peculiarly distressing disease, let us examine the other patient whom I have brought before you. I am indebted to Dr. Beevor's notes for many particulars of his case.

James H. is 64 years of age. He has been an upholsterer's foreman, has worked hard, and, according to his own account, has always been steady. He has been married 30 years, has had four children, and his wife has never miscarried. There is no history of phthisis, insanity, or fits in his family, but a doubtful account of hemiplegia in his mother. He says that he never had scarlet fever or rheumatic fever, or syphilis. He had "typhus" fever when young, and has suffered a little from gravel. His present illness dates from only two years.

At first he found difficulty in using his knife and fork and in putting on his clothes. About four months later he began to drag his feet in walking, and gradually power was lost more and more completely in all his limbs. Six months ago he began to "speak thick," according to his own description, and this has since become worse. He has not had trouble in masticating, and could always protrude his tongue. He says that he has sometimes choked in swallowing, but no difficulty has been observed in this respect since his admission into hospital. For six months past he has noticed that when his arms were raised they remained in the acquired position longer than was natural. He has not been able to walk for a year past, or even to get out of bed. His legs and arms have been stiff for the last nine months.

The patient is a very stout old man, who constantly lies in bed without occupying himself in any way. He cannot help himself at all. There is an aspect of marked mental hebetude, or at all events an extreme slowness of expression, so that it is difficult to elicit answers to questions about his history. The difficulty is partly also attendant upon his mumbling speech, which is sometimes quite unintelligible. Yet we contrive to get, in process of time, though the task is laborious, a fair amount of information from him. The face wears a peculiarly stolid expression. When told to show his teeth he makes only a slow and feeble attempt; he can close his eyes,

but cannot screw his eyelids up tight. The tongue is protruded straight although slowly, and it does not show any tremor. Cutaneous sensibility is nowhere affected. The patient has not suffered from pains in his head or body. The movements of the eye-muscles are natural. There is no nystagmus.

The upper extremities are thin in proportion to the lower, but the emaciation, if so it can be called, is symmetrical on the two sides, as is shown by the following measurements. Right forearm, $8\frac{3}{4}$ inches; left forearm, $8\frac{1}{2}$ inches. Right arm, $9\frac{1}{2}$ inches; left arm, $9\frac{1}{4}$ inches. The hands remain in a position of semiflexion, the fourth and fifth fingers more flexed than the second and third. The finger-portion of the hand is much drawn over to the ulnar side—the thumb is applied against the forefinger. The attitude of the hands is exactly that of Paralysis agitans, but there is no tremor whatever in the fingers. The two distal phalanges appear somewhat over-extended upon the proximal phalanx. The intrinsic muscles of the thumb and the interossei appear to be thin.

As you observe, this man can flex and extend his fingers, but the movements are exceedingly slow. He can, in the same deliberate way, flex and extend (though not to the full extent) both elbow-joints, but there is very little capacity for pronation or supination. He can raise his arms straight up into the air and abduct them from his trunk, the shoulder-joints being less affected than the elbows.

There is a very curious circumstance to be noted in connection with the movements of his limbs. In response to my request, he raises his hand or his foot to some distance above his couch, and there he lets it remain for several seconds until, indeed, he is told to drop it; and if I lift one of his limbs, and place it in any position, so it remains in a cataleptic fashion. When told to replace it, it is long before he makes any movement, and then the limb descends slowly. The muscles of his trunk appear to be weak; he cannot raise himself in bed, and, indeed, does not move at all—lying, if not disturbed, for hours together in the position in which he is placed.

As regards the lower limbs, they are not so much emaciated as the upper extremities, but the muscles are flabby. He can

lift either heel very slowly off the couch, can bend either knee, but can only move the ankle-joints a very little. All the joints appear very stiff, and require considerable force to move them.

The patellar tendon-reflex is only just obtained. There is no ankle-clonus. The cutaneous reflex of the foot-sole is obtained on each side.

The right pupil is smaller than the left. They both react to light.

The heart-sounds appear distant; there is no murmur. As regards his mental state, in reply to inquiry he says that "he feels lost." He says "he wants to give his address," and gives 139 Bow Road, which is not altogether correct. He seems very obtuse, and scarcely speaks at all, though he is always apparently conscious.

The faradaic excitability of the muscles of the forearms is normal, that of the arms and lower extremities exhibits a very slight and unimportant lowering.

The action of the rectum and bladder is preserved. He has no difficulty in retaining his urine or fæces, but sometimes a little delay occurs in expelling the contents of the bladder.

There is no disposition towards the formation of bed-sores.

The ophthalmoscope shows no change in the fundus oculi.

These represent the principal symptoms of a condition which is manifestly of an unusual and obscure character. I have been led to conclude that the patient represents an anomalous form of Paralysis agitans, partly by the necessity of excluding other explanations, and partly by the resemblance of the patient's state to that which would be produced by an intensification of certain symptoms of shaking palsy. Let us first see how far his condition can be made to tally with that produced by certain other conditions.

The preserved electric irritability of the muscles enables us at once to exclude, what otherwise might be a not unlikely supposition, that the patient is suffering from a severe form of lead-poisoning. In such a condition the faradaic excitability would be very much reduced, or, still more probably, absent. Progressive muscular atrophy is excluded by the universal character of the lesion. As you are aware, that disease picks

out groups of muscles physiologically associated in their action, leaving others intact. Here the affection is a general one of the whole muscular system, and, in addition to this, the mental condition, or, at least, the laborious slowness of movement in response to order which we observe in this man, forms no part of the history of progressive muscular atrophy. Bilateral sclerosis, which at first suggests itself, on account of the rigidity of the limbs, is excluded by the condition of the tendon-reflexes, which in that disease would certainly be exaggerated. So also would they be, though not to an equal extent, in Charcot's amyotrophic lateral sclerosis. In both these conditions, the one dependent on sclerosis of the lateral columns alone, and the other upon sclerosis of those columns along with lesion of the large ganglionic cells in the anterior cornua, the tendon-reflexes are intensified. For the same reason, cerebro-spinal insular sclerosis must also be excluded.

On the other hand, let us see what symptoms are to be found which accord with the view that the patient is affected with Paralysis agitans. We have not here, it must be remembered, the opportunity of seeing him walk. That is a great loss, for the attitude of a patient with shaking palsy is, as you have seen, exceedingly characteristic. "The patient," as Charcot says, "loses the faculty of preserving equilibrium whilst walking. In some we notice a tendency to propulsion or to retropulsion;" (in this female, as I showed just now, the tendency to retropulsion when I pulled the skirt of her dress is well marked) "without feeling any giddiness, the patient is in the first case propelled forward and, as it were, compelled to adopt a quick pace; the individual is unable without extreme difficulty to stop—being apparently forced to follow a flying centre of gravity." He adds: "A peculiar attitude of the body and its members, a fixed look, and immobile features should also be enumerated among the more important symptoms of this disease." Again: "The muscles of the face are motionless, there is even a remarkable fixity of look, and the features present a permanent expression of mournfulness, sometimes of stolidness or stupidity."

Although we cannot see this patient stand or walk we have the opportunity of noting the fixed look and immobile

features which singularly resemble this description, as well as what we observe in the other patient. Might not a widespread softening of the brain be attended with a similar physiognomy? I think it might. But it must be remembered that the mode of progress of the disease in this man does not accord with such a supposition. There is a gradually progressive loss of power and increasing rigidity in his members. When you have widespread atheromatous changes in cerebral arteries leading to thrombosis and softening, the paralysis which ensues, although it may easily involve all the limbs, does so not by slow and imperceptible degrees, but by leaps. A more or less sudden loss of power occurs in one limb, and after an interval of varying duration the same thing happens in regard to another. Moreover in such a case again we should certainly expect the tendon-reflexes to be exaggerated —not lowered in activity as is the case in this man's knees. The same argument applies against the suggestion of the growth of a cerebral tumour, which besides is additionally rendered improbable, though not, it must be allowed, impossible by the absence at any time of pain in the head, and vomiting, and the fact that no changes are discernible with the ophthalmoscope. But there are still two other circumstances which, although not absolutely conclusive, speak most strongly against the view that the condition described depends on a destructive intracranial lesion. The patient is able to retain his urine and fæces. If his helplessness were dependent upon extensive brain softening, it is in the highest degree improbable that the power over the bladder and rectum would have been maintained. I mention this as a result of clinical experience without being able to say what portion of the encephalic centres must be involved to induce this particular loss of power. It is a fact that in more or less generalised paralysis from cerebral softening we do constantly find that the evacuations cannot be restrained.

The second circumstance is that the patient, though he lies in the position in which he is placed, shows no disposition to the formation of bed-sores. It is true that he was placed on a water-bed as soon as he arrived, but the helplessness which we see had existed for months before his admission into hospital,

and during that time he did not enjoy this advantage. But besides the expressionless features and fixity of looks, we have here two other symptoms of Paralysis agitans of great importance. The first is the attitude of the fingers which, as you have seen, precisely resembles that which is to be observed in the female patient. Now this attitude is very peculiar. It does not occur in ordinary cases of hemiplegia or generalised paralysis from extensive cerebral softening. In such cases it is true the hand tends to assume a position of more or less strongly marked flexion, but *the fingers are also and indeed especially flexed.* Here on the contrary the fore-finger and middle finger at least tend rather towards an attitude of over-extension. The phalangeal portion of the hand is besides inclined towards the ulnar side. It was the look of these hands which first suggested to me the idea that the patient might be suffering from Paralysis agitans.

Another symptom is the rigidity. A patient of Charcot's said that his joints appeared "soldered together." Benedikt suggests that the habitual rigidity of a certain number of the muscles undoubtedly contributes in rendering movement laborious.

The muscular rigidity which occurs in Paralysis agitans seems to me peculiar. It is not apparently a spastic condition like that e.g. which we see in the late rigidity of hemiplegia or in the sequel of myelitis with secondary degeneration of the lateral columns of the cord. It resembles to a remarkable extent the stiffness which results from disuse. You see this condition well marked in the muscles of a forearm which has been for some weeks confined to a splint on account of fracture. The muscles are feeble and stiff, but the stiffness can be overcome with tolerable ease by passive movements. There is no tension of the muscles in this case any more than there is in the case of the fractured arm, and here, as you see, they are actually flabby. Along with his rigidity, if such it can be called, is a condition which Charcot has called attention to —a retardation in the execution of movements. The patient performs movements with extreme slowness. "In relation to the faculty of speech," Charcot writes, "there is a comparatively considerable lapse of time between the thought and the act.

One might suppose that the nervous influx cannot be set to work until after extraordinary efforts." Probably, also, I would suggest, the faculty of receiving impressions is likewise defective.

The muscular stiffness is commonly most marked in the advanced stages of the disease. Charcot writes: "There are cases, though these are rare indeed, in which muscular rigidity is a symptom of the early stage of the disease, and a really prominent one. I have recently observed an example which belongs to this category. The patient had scarcely noticed the tremor, which, in fact, showed little intensity in his case, and was confined to one hand. He already displayed, however, in a high degree, the peculiar attitude of the body and its members, the difficulty of movement and the characteristic gait." In a case under the care of Dr. Gowers in this hospital, the patient, a female, exhibited all the symptoms of Paralysis agitans except the trembling, which was barely perceptible. In this man there is an entire absence of tremor. But we do not know whether the symptom has always been absent. It is quite possible that there may have been a certain amount, perhaps too small to attract the patient's attention. However that may be, I do not consider that the total absence of trembling is any bar to our reception of this case as one of Paralysis agitans. The remaining symptoms appear to me to be quite sufficient to enable us to relegate the case to that category. I may remind you of the analogy in this respect with what obtains in another disease of the cerebro-spinal nervous system—tabes dorsalis. The cases are numerous in which the symptom ataxy of gait which gives the name progressive locomotor ataxy to the disease, is entirely absent.

It is very probable that such cases as this man's are not so rare as might be thought. Looking back to past experience, I am disposed to think that I have seen several such in workhouses and hospitals, where they have been classed with cases of softening of the brain.

But the converse to this total absence of tremulousness may be observed. The movements may be so intensified as to occupy almost the whole attention. I lately saw the following case:

Mrs. M., 64, a farmer's widow (April 26, 1881). Twenty-seven years ago she began to get weak in the right hand, and thirteen years since her head began to shake.

Now as she sits she is constantly moving all over; her head is jerked, the arms, legs, and body writhing. She is constantly gasping in her breath, her face twitching—in an incessant state of distress. The larynx is prominent, owing to great action of the sterno-mastoids and trapezii. There is, she says, a "distressing feeling in her inside."

She is constantly obliged to be occupying herself. Whilst I examined her she was knitting.

Her daughter says she is better in herself than she was six years ago, but the movements are about the same.

She sleeps about four hours at night.

The general health is good.

She is "never ill." She always feels better when by herself. Any nervous excitement increases her distress.

There is constant snorting and sniffing.

Here is an example of another variety:

William W., æt. 64, was admitted into the hospital on May 20, 1878, with Paralysis agitans, which had begun five years previously.

He was an engine-driver who had never had a day's illness in his life. In the course of his work he had to crawl into very hot boilers to clean them out, and to this he attributed his illness.

His symptoms began with subjective coldness of the knees, shortly followed by shaking of the left hand, left leg, and then of the right hand.

There was almost constant shaking of the left hand. If he took hold of an object and held it firmly, the shaking stopped for a few seconds, but then recommenced worse than ever.

In this man there was a very peculiar condition. Whilst he was at rest there was no shaking of his legs, but if he stood up and attempted to walk, he remained for a time unable to start, his feet, however, beating the ground rapidly. All of a sudden this would cease, and he would start off at a fair pace, though he required some one to hold him up. This "marking time'

action when he tried to walk had been observed about eight months. The patient, who was an engine-driver, appeared much struck with Mr. Broster's suggestion, that it reminded one of the wheels of a locomotive failing to bite the rails when they are slippery with frost, and making, in consequence, ineffective revolutions.

Paralysis agitans is sometimes acute in its progress, and this, I think, is more often the case when the patient is comparatively young. I lately saw a man, æt. 38, whose symptoms dated from one year only. They had commenced with hesitation of speech, which was followed by shaking, first of the left and then of the right arm, and when he came to me he presented all the symptoms in a marked degree.

There was a patient in the hospital, a year or two ago, who was still younger. I gather the following account from Mr. Broster's notes, taken on his admission:

Benjamin L., æt. 22, was admitted into hospital on April 30, 1879. His occupation is that of a butcher, and for five years past he has been in London, having previously lived in the country. He looks about his age. Somewhat apathetic in manner, he yet complained of irritability of temper, especially when exposed to noise. His memory is good. He has no headache nor giddiness. The speech is thick, and somewhat indistinct; the articulation being hurried, and the words run together. The tongue showed marked tremor. Saliva dribbled from his mouth at times. There is difficulty in swallowing, and he is obliged to have his food chopped up fine.

His face wears a fixed look. He cannot frown, and can only show his teeth feebly by voluntary movement. During involuntary movement, on the other hand, as when he laughs, he shows them very well. He has no emotional instability. As he sits, there is marked shaking of the head and upper limbs. The arms—especially the right arm—are in a continual state of rather violent agitation, which is increased by excitement, and is least when he is left quietly to himself. There are also involuntary uniform movements of the legs and feet when he is seated. If he lifts his heel off the ground, so as to rest solely on the ball of the foot, there is a rapid movement similar to foot-clonus. No foot-clonus can be elicited

by sudden passive dorsal flexion of the foot. The patellar tendon-reflex is normal—free from exaggeration. In his walk he inclines forward, and "scuffles" along, but not so hurriedly as is often seen in these cases. He complains of stiffness in his arms and legs.

When he picks up anything with either hand there is a momentary lull in the movement, and he takes hold of them quietly and steadily. Immediately afterwards the movements recommence. He is very easily pushed forwards or backwards, however much he may try to resist this.

His breathing is of a half-sobbing character.

There is slight lateral curvature. No spinal tenderness.

Now, in this case, it was only eleven months before these notes were taken that the patient had first noticed a slight shaking of the right hand. This very gradually increased, and in three months he was forced to give up his occupation. Two months later he noticed that his left hand was beginning to shake like the right, and after two or three months more his legs also became involved. There was shaking whilst he sat, and he says that they shook all the more if he bore his weight upon his toes. A month or two after this, his swallowing became affected, and his mouth, according to his account, was always full of saliva. About the same time his articulation became embarrassed.

This man acknowledged a course of life of the most dissipated character. He had lived freely and drunk heavily. From the age of 16 to 21 he had masturbated two or three times a day; and from 21 to 22, about the time of the commencement of his symptoms, had indulged to the greatest excess in sexual intercourse. He had never had syphilis, but had once suffered from gonorrhœa. The family history was a healthy one.

It is worth noting that, in the other case—patient aged 38—there was a history of excessive sexual intercourse immediately preceding the commencement of the symptoms.

The patient, William V., who is now attending the hospital, is affected with shaking palsy, which commenced in his left arm. By the dynamometer, the grasp of the right hand measures 50, that of the left 40.

The movements in this case are of a somewhat unusual

character. They are much larger in extent than is commonly seen, so that as the patient sits he flaps his knees with both hands noisily and roughly. His legs also are affected with coarse movements. His walk is characteristic, the body being bent forward, and the head maintaining a "set" position.

If we ask him to pick up a pen off the table, his arm is still whilst he is stretching it out to grasp the object; but when the hand closes upon it the movements begin to recur with great energy.

Another of our out-patients, a female, presents symptoms which are at first strongly suggestive of this disease, but I do not think she is an example of it.

Elizabeth B. is a widow, 66 years of age, who was strong and well, and had nothing to complain of till ten weeks ago, when she woke up one morning retching and vomiting, and found that she had lost power to a certain extent in the left arm and leg. She found that she could not keep her arm still, and her leg also was constantly shaking. A very aged neighbour who came to her, said, "Don't touch me, or you'll pull me down," the shaking was so violent. She knocked the left knee against the right so violently that it was quite bruised. She remained in bed three weeks, during which the movements were very violent, but afterwards they became less so.

Now, we observe that as she rests her hand on her knee it is steady, but when she tries to pick up things from the table there are rhythmical movements—flexion and extension movements of the hand numbering about 180 in the minute. The attitude of the hand is not that of Paralysis agitans. A friend who comes with her says that she is sometimes obliged to throw the bed-clothes off, she complains so much of heat. Since she has been here before us, her face has come over in a red flush several times.

The wrist-reflex is exaggerated on both sides, but especially on the left, whilst the patellar tendon-reflex approaches to a clonus on the left, and is in some excess also on the right side. She walks with short toddling steps, and is compelled to use a stick, otherwise she falls. The left knee never shakes now, but

sometimes in walking it "catches up." The dynamometer shows a grasp of 40° with the right, 22° with the left hand.

She has never had any numbness in the left hand, but there is often aching pain in the forearm, especially towards night.

We do now and then observe a state of tremor in the paralysed limbs of a hemiplegic patient; but so far as I have seen, it is very rare to find such an amount of tremulous movement as we see in this woman. Here the tremors closely resemble those of Paralysis agitans, and the case might easily be mistaken for one of that disease.

According to Nothnagel, the condition has been observed in connection with lesion in the following localities:—1. The internal capsule in its posterior segment. 2. Optic thalamus. 3. The foot of the corona radiata. In these situations it is, he points out, that lesions are most apt to be accompanied by hemichorea and athetosis, with which it is supposed these tremors are nearly allied. It is not certain whether the condition occurs as a result of cortical lesions.

This patient evidently had a sudden cerebral attack, accompanied by vomiting and loss of power, in the arm and leg of the left side. In the suddenness of its origin, the disease contrasts as strongly as possible with shaking palsy.

The exaggeration of reflex amounting to foot-clonus on the affected side, is also opposed to the diagnosis of that disorder. The attack has, probably, been of an apoplectic character, the exact situation of which, however, I am not prepared to indicate with any confidence.

Passing over the morbid anatomy of Paralysis agitans, about which nothing conclusive is as yet established, I would refer to one or two points in regard to the symptoms.

If we examine the upper extremity in a case of true shaking palsy, we are struck at once by the circumstance that apparently the small muscles, those concerned in the most delicate movements, are those which suffer most. The attitude of the fingers is that which would be produced by alternate contraction and relaxation of the fibres of the interosseous muscles. It is unusual in other conditions to find predominance of affection of such muscles over the larger ones. In the climax of an epileptic seizure the hand is clenched.

The contraction of the large and powerful flexor muscles lying on the anterior surface of the forearm, overpowers completely that of the small interossei. So also in the converse of convulsion—in hemiplegia, when the paralysis is complete the fingers lie passively in an attitude of semiflexion. There is only one condition, I think, in which the position of the fingers at all resembles that which they occupy in shaking palsy, and that is the singular disorder called Tetany. It is not easy to see any kind of association between these disorders, but the point is perhaps worth noting.

As regards the piping senile voice, to which I have already referred, I have lately met with an instance in private practice which has afforded me an opportunity of investigation. The patient, William B., aged 57, has the characteristic fixed look and the piping voice. His right hand, ever and anon, but not constantly, is shaken to and fro. The left also is shaken, but much less. He feels very weak in the right arm, and says that he has scarcely any use at all in it.

He does not come over in flushes of heat or perspiration.

Sometimes, he says, he can scarcely talk. This, he thinks, is owing to something in the throat. His wife has noticed the alteration in the voice.

About two years ago he began to feel loss of power in the right arm—that hand would drop occasionally from the wrist so that he could not depend upon holding things. The loss of power has gradually increased, especially of late. Within the last few weeks he has inclined to trip with the right leg, which feels, as it were, shorter than the other.

Lately, for a fortnight past, he has been liable to giddiness, and on one occasion he nearly fell down.

He does not suffer from sickness. He has usually had good health. His appetite is good; he sleeps well; the bowels act.

He has a difficulty in holding his urine, and would wet himself if he were not very quick. He gets up two or three times in the night to pass water—just a little.

Memory is good. He would rather be by himself, and avoids noise and company. There is a fixed look of the face with half a frown upon it, and the head is held stiffly, the eyes usually looking straight in front.

The tendon-reflex at the wrist is equal on both sides, and not abnormal.

At my request Dr. Felix Semon was good enough to examine this patient with the laryngoscope. His report is that there is neither motor nor sensory paralysis nor loss of reflex-sensibility to be detected in either pharynx or larynx, and that both these parts are quite free from traces of previous specific or other disease. It seems probable, therefore, that the high tone, the piping voice, which I have likened to that adopted by an actor who represents advanced age upon the stage, may be dependent upon a functional imperfection of the soft palate, perhaps due to a vibratile condition of its muscular structure.

DEAN SWIFT'S DISEASE.

BY DR. BUCKNILL, F.R.S.

DURING the past autumn I received a letter from a gentleman engaged in literary work, requesting my opinion on the "mysterious disease" of the great author and wit, whose name distinguishes this paper. My interlocutor particularly wished to know whether the sane part of Swift's life was likely to have been in any way affected by the latent presence of insanity; whether a correct diagnosis was possible; whether parallel cases were on record; and, finally, whether a surfeit of green fruit, at the age of twenty-three years, was capable of resulting in the absolute fatuity from which the patient suffered at seventy-five.

This questioning has stimulated me to an investigation which I had thought was already threadbare, but which I found full of interest; and when I say that, upon weighing the evidence, it will probably be acknowledged that Jonathan Swift's mysterious disease was an instance of that curious form of disease, Labyrinthine Vertigo, or Le Maladie de Ménière, the knowledge of which is one of the most recent triumphs of pathological research directed by physiological experiment, it will scarcely be thought that it was needless to reopen a controversy in which already everything had been said which ought to have been said, and not a little which ought not to have been said.

When Dr. Beddoes suggested that Swift's ailments and his conduct towards women were due to dissolute habits in youth, Sir Walter Scott replied, that "until medical authors can clearly account for and radically cure the diseases of their contemporary patients, they may readily be excused from assigning dishonourable causes for the disorders of the illustrious

dead." But if Dr. Beddoes were unquestionably wrong in making such a suggestion without evidence, Sir Walter was scarcely right in making his retort too general; for if medical opinions respecting the states of mind of persons who have departed this life must be forbidden until medical men can ensure the radical cure of diseases, not only will much valuable evidence respecting the validity of wills be excluded, but the science of pathology itself, depending upon the history of diseases and verified by observations made after death, must be interrupted until an event which seems impossible has taken place.

Whether the causes of disease are or are not dishonourable, and whether the subjects of them are or are not illustrious, has nothing to do with the scientific question; and the often-quoted sneer of Swift's greatest biographer at the medical profession seems, when examined, as silly as general sarcasm usually is. Undeterred by such sarcasm, an eminent medical man did investigate the causes for the disorders of the illustrious dead in a work which he modestly called an Essay, published in 1849, and entitled 'The Closing Years of Dean Swift's Life,' &c., by W. R. Wilde, M.R.I.A., F.R.C.S. This little work, marked by the excellencies of careful research, sound reasoning, moderate opinions and fair conclusions, would have rendered further discussion needless if medical science had stood still since its publication; but the advances made in medical psychology during the last thirty-two years might give us some excuse for reconsideration even if Ménière had never made his discovery of a definite form of disease previously unrecognised, which appears to conform in all important points with the lifelong disease of the illustrious Dean.

Even in Swift's latest biography, or rather that fragment of one which so strongly makes us feel that touch of the vanished hand of the most appreciative critic of men of letters, the late John Forster's charming volume, the author speaks of Swift's giddiness and deafness, not as symptoms of one disorder, but as "two lifelong enemies," and it is a curious enough fact that Swift himself attributed the origin of these two enemies to different causes, operating at different periods. In a letter to

Mrs. Howard in 1727 he writes: "About two hours before you were born I got my *giddiness* by eating a hundred golden pippins at a time at Richmond; and when you were four years and a quarter old, bating two days, having made a fine seat further in Surrey, where I used to read, there I got my *deafness*, and these two friends, one or other, have visited me one or other every year since; and, being old acquaintances, have now thought fit to come together." Mrs. Howard having been born in 1690, the date of the deafness given in this letter would be 1694, when Swift was twenty-seven years old. But in a passage quoted by Forster, p. 48, Swift wrote, "In England before I was twenty I got a cold, which gave me a deafness that I could never clear myself of .... my left ear has never been well since."—April 30, 1737.

One cannot but concur in Johnson's remark on the above, that "The original of diseases is commonly obscure, and almost every schoolboy eats as much fruit as he can get, without inconvenience." But it may also be remarked that if Swift had been "a contemporary patient," although we might not have effected a radical cure of his disease we should at least have understood enough of its origin and nature to have saved him from tormenting himself by a lifelong abstinence from fruit, of which he was passionately fond, under the belief that it had caused and continued to excite his disease by inducing that "coldness of stomach to which he attributed his vertigo and its accompanying sickness."

In a letter of 1708 he says that, "I was through a long time pursued by a cruel illness that seized me at fits and hindered me from pursuing any business." It is possible that this illness was but a return of the dangerous colic from which he suffered in 1696; and it is not until 1710 to 1713, and while residing in London, that he describes in some detail the symptoms of his lifelong complaint in his 'Journal to Stella.' The most descriptive passage is perhaps the one dated Oct. 31, 1710:—

"This morning, sitting in my bed, I had a fit of *giddiness*; the room turned round for about a minute, and then it went off, leaving me *sickish*, but not very. I saw Dr. Cockburn to-day, and he promises to send me the pills that did me good

last year; and likewise has promised me an oil for my *ears*, that he has been making for that ailment for somebody else."

A fit of giddiness, with sickness and ear-disease, is not this labyrinthine vertigo?

On different days in *Jan.* 1711 he writes:—

"I had an ugly fit in my chamber last night." . . . "My head is not in order, and yet is not absolutely ill; but giddyish, and makes me listless." . . . "One fit shakes me a long time."

Feb. 1.—"I walked into the City to dine, but I walked plaguy carefully, for fear of sliding against my will."

April 18.—"I did not go to the House of Commons about the yarn: my head was not well enough. I know not what is the matter. It has never been thus before; two days together giddy from morning till night, and I totter a little, but can make a shift to walk."

In *May*: "I do not totter as I did, but walk firm as a rock, only once or twice for a minute."

Sept. 1*st* he notes an important peculiarity, distinguishing cerebral from stomachic vertigo:—"My head is pretty well; only a sudden turn at any time makes me feel giddy for a moment, and sometimes it feels very stuffed."

The journals of October show that he distinguished ordinary from vertiginous headache:—"My head has ached a little in the evening, but it is not of the true giddy sort, so I do not much value it." . . . "I had a little turn in my head this morning, which, though it did not last above a minute, yet being of the true sort, has made me as weak as a dog all this day."

During the years of residence in London which embrace the period of the 'Journal to Stella,' his other enemy, deafness, is only referred to incidentally, as when he compares it to that of the Lord Treasurer; but after his return to Ireland, his deafness becomes sufficiently severe to make him complain.

In 1720 he writes:—"What if I should add that once in five or six weeks I am deaf for three or four days."

In 1724 he writes:—"I have been this month past so pestered with a return of the *noise and deafness* in my ears that I had not the spirit to perform the common offices of life." Subse-

quently, in the same year: "My deafness has left me above three weeks, and therefore I expect a visit from it soon." It was evidently periodic and paroxysmal, like the giddiness.

He complains in another letter of an old vexatious disorder of a deafness and *noise* in the ears. In 1827, in a letter to Sheridan, he says that his deafness is worse than it ever before had been, and that it is accompanied by giddiness and tottering. "I believe," says he, "that this giddiness is the disorder which will at last get the better of me." And again, "I walk like a drunken man, and am deafer than ever you knew me."

In 1728, in "about eight months," says Wilde, "he had half-a-dozen attacks of the giddiness and sickness, each of which lasted about three weeks." But in 1731 he wrote to Mr. Gay, "The giddiness I was subject to, instead of coming seldom and violent, now constantly attends me more or less, though in a more peaceable manner, yet such as will not qualify me to live among the young and healthy." In 1736, writing to Pope, "years and infirmities have quite broke me. I mean that *continual* disorder in my head." In 1737, to Alderman Barker, "I am forced to tell you my health is much decayed; my deafness and giddiness more frequent; spirits I have none left; my memory is almost gone."

Long before, however, these symptoms had commenced. Impairment of memory he complained of as early as 1713, after the attack of shingles; and later on in the same year he speaks of his horrible melancholy changing into dulness, and from thenceforth increasing irritability of temper and mental depression are traceable throughout his history and correspondence. Not that he was at any time really of unsound mind or incapable; for when in 1737, in the Bettesworth affair, a gratifying address was presented to him, it is recorded that "when this paper was delivered Swift was in bed, giddy and deaf, having been some time before seized with one of his fits; but he dictated an answer in which there is all the dignity of habitual pre-eminence and all the resignation of humble piety."

The above quotations are but a selection from a far greater number of references which might be made to Swift's letters

and journals, affording conclusive evidence, as I venture to think, that he suffered from twenty years of age from the disease, whose characteristic symptoms are, "that the patient is suddenly seized with vertigo and a feeling of nausea or positive sickness, with great constitutional depression and faintness. Usually the giddiness comes on simultaneously with ringing or buzzing in one or, it may be, both ears."—*Ferrier.*

It has this year been pointed out by Féré, in the 'Revue de Médecine,' that there are two forms of the disease to be recognised, "une forme grave avec état vertigineux à peu près permanent interrompu par les paroxysmes, et une forme moins fâcheuse, constituée par des accès séparés par des périodes de santé parfaite." "Dans la forme bénigne [of which Swift's was an example] les accès ne se produisent quelquefois qu'à des distances très éloignées. E. Ménière cite une malade qui eut une rémission de onze mois. Pendant ces périodes d'accalmie, la surdité persiste avec une intensité variable, et elle s'accompagne souvent des sensations subjectives intermittentes de l'ouïe. La maladie elle-même dure tant que la surdité n'est pas absolue."

Up to the date to which we have traced the progress of the disease, it appears to have been purely a physical malady, with no mental symptoms, unless some degree of loss of memory can be so called. Swift, indeed, complains bitterly of the impairment, as if memory were gone, and in his declining years of age and sickness it may have been a dull function compared with the brilliant faculty he once possessed. But clearly the memory was still serviceable which enabled him to compose, with wonderful vivacity, even such poetry as that outburst against political and social corruption—'The Legion Club'—which Jeffrey thinks "deserving of attention as the most thoroughly animated, fierce and energetic of all Swift's metrical compositions; and though the animation be altogether of a ferocious character, and seems occasionally to verge upon absolute insanity, there is still a force and a terror about it which redeems it from ridicule, and makes us shudder at the sort of demoniacal inspiration with which the malison is vented." This poem, written in 1736, was his last work; its appreciation by his most hostile critic will show how little he suffered from loss of any mental faculty when he wrote it.

That disease and grief had made him irritable and passionate, and often desponding, is clear enough from his correspondence and the accounts of him which have come down to us; but that there was any failure of mind this 'Legion Club' fully disproves; and if fiercely expressed hatred is any evidence that an author is on the verge of insanity, Jeffrey must have been curiously insensible to the testimony he was bearing against his own soundness of mind in his criticism of the greater master of his own art.

Between 1736, when Swift wrote 'The Legion Club,' and 1741, when the outbreak of insanity really took place, there can be no doubt that he passed through a period of great wretchedness and depression—he was "miserably ill." He had lost to a great extent two of his senses, for he was deaf and his eyesight failed; his dearest friends had died before him, and his public sympathies were constantly outraged.

In 1738 he wrote to Alderman Barker, "I have for almost three years past been only the shadow of my former self, with years of sickness and rage against all public proceedings, especially in this miserably oppressed country. I have entirely lost my memory, except when it is aroused by perpetual subjects of vexation."

Two years later he wrote the following pathetic letter to his old friend Mrs. Whiteway:—

"I have been very miserable all night, and to-day extremely deaf and full of pain. I am so stupid and confounded that I cannot express the mortification I am under, both of body and mind. All I can say is that I am not in torture, but I daily and hourly expect it. Pray let me know how your health is, and your family. I hardly understand one word I write. I am sure my days will be very few; few and miserable they must be. I am for these few days yours entirely,

"J. Swift."

"If I do not blunder it is Saturday."

A very pitiful state this period of becoming insane, and yet not having become so. But even at this late date one cannot recognise the invasion of mental disease. Misery and despondency there was, more than enough, but not madness, unless Job was mad. But Swift was rapidly tending towards

madness, and he knew it, for strong forebodings of insanity, which are not common, existed in him in a remarkable degree. Sir Walter Scott says that "his first state was that of violent and furious lunacy;" but Sir William Wilde points out that all the biographers have had no other sources of information as to the outbreak and history of his insanity than two letters; one from Mrs. Whiteway, and one from his cousin, Mr. Deane Swift.

Mrs. Whiteway wrote Nov. 22, 1742, as follows, omitting some expressions of feeling:—

"I was the last person whom he knew, and when that part of his memory failed he was so outrageous at seeing anybody that I was forced to leave him, nor could he rest for a night or two after seeing any person. He walked ten hours a day, would not eat or drink if his servant stayed in the room. His meal was served ready cut, and sometimes it would be an hour on the table before he would touch it, and then eat it walking. About six weeks ago, in one night's time his left eye swelled as large as an egg, and the lid, Mr. Nicholls [his surgeon] thought would mortify, and many large boils appeared on his arms and body. The torture he was in is not to be described. Five persons could scarce hold him, for a week, from tearing out his own eyes; and for near a month he did not sleep two hours in twenty-four. Yet a moderate appetite continued; and, what is more to be wondered at, the last day of his illness he knew me perfectly well, took me by the hand, called me by my name, and showed the same pleasure as usual in seeing me. I asked him if he would give me a dinner? He said, 'To be sure, my old friend.' Thus he continued that day, and knew the doctor and surgeon and all his family, so well that Mr. Nicholls thought it possible he might return to a share of understanding, so as to be able to call for what he wanted, and to bear some of his old friends to amuse him. But alas! this pleasure to me was but of short duration; for the next day or two it was all over, and proved to be only pain that had roused him. He is now free from torture, his eye almost well, very quiet, and begins to sleep, but cannot without great difficulty be prevailed upon to walk a turn about his room; and yet in this way, the physicians think, he may hold out for some time."

The only other authentic account from personal knowledge is contained in the letter of Mr. Deane Swift to Lord Orrery, dated April 4th, 1744. After stating that a thousand stories of the illness had been invented and imposed upon the world, he proceeds to state some facts witnessed by himself.

"On Sunday the 17th of March, as he sat in his chair, upon the housekeeper removing a knife from him as he was going to catch at it, he shrugged his shoulders, and rocking himself, he said, 'I am what I am. I am what I am;' and about six minutes afterwards repeated the same words two or three times over. Sometimes he will not utter a syllable, at other times he will speak incoherent words; but he never yet, as far as I could hear, talked nonsense, or said a foolish thing. About four months ago he gave me great trouble. He seemed to have a mind to talk to me. In order to try what he would say, I told him I came to dine with him, and immediately his housekeeper, Mrs. Ridgeway, said, 'Won't you give Mr. Swift a glass of wine, sir?' he shrugged his shoulders, just as he used to do when he had a mind that a friend should not spend the evening with him. Shrugging his shoulders, your Lordship may remember, was as much as to say, 'you'll ruin me in wine.' I own I was scarce able to bear the sight. Soon after he again endeavoured, with a good deal of pain, to find words to speak to me; at last, not being able, after many efforts, he gave a heavy sigh, and I think was afterwards silent. This puts me in mind of what he said about five days ago. He endeavoured several times to speak to his servant [now and then he calls him by his name]; at last, not finding words to express what he would be at, he said, 'I am a fool.' Not long ago the servant took up his watch that lay upon the table, to see what o'clock it was; he said 'bring it here,' and when it was brought, he looked very attentively at it. Some time ago the servant was breaking a large stubborn coal, he said; 'that's a stone, you blockhead.' In a few days, or some very short time after guardians had been appointed for him, I went into his dining-room, where he was walking; I said something to him very insignificant, I know not what, but, instead of making any kind of answer to it, he said, 'Go, go,' pointing with his hand to the door, and immediately afterwards, raising his hand

to his head, he said, 'My best understanding,' and so broke off abruptly, and walked away."

These two letters are stated by Sir William Wilde to be the only account of the last three years of Swift's life that has come down to us. He died October 19th, 1745, in the seventy-eighth year of his age. His death, according to Lord Orrery, being easy, without the least pang or convulsion; but according to Faulkner, being one of "great agony, with strong convulsive fits" for thirty-six hours before. The only record of the autopsy which was made is that Mr. Whiteway "opened the skull, and found much water in the brain." A more interesting record, however, remains in the plaster cast of Swift's head. Of this Sir Walter Scott says that "the expression is most unequivocally maniacal, and one side of the mouth horribly contorted downwards as if in pain." But Sir William Wilde, whose observation we greatly prefer in such a matter, says, "The expression is remarkably placid, but there is an evident drag on the left side of the mouth, exhibiting a paralysis of the facial muscles to the right side. Upon the back of the cast are two lines of writing, greatly defaced, of which this much can still be read, 'Dean Swift, taken off his * * * * the night of his burial, and the f * * * * one side larger than the other in nature * * Opened before * * * The mould is in pieces.'" A deep indentation, says Wilde, shows where the calvarium had been sawn; and accurately corresponds with the division of the skull found in Swift's coffin in 1835. On the same excellent authority we know that the cast of the interior of Swift's skull is remarkable as showing the enormous development of the vessels within the cranium, the very small anterior lobes, the great size of the glandulæ pacchioniæ, the exceedingly small cerebellum, a natural formation as may be seen by the very low position of the tentorium, the immense size of the posterior and middle lobes, particularly the former, and the absence of any appearance of disease in the anterior lobes, as far at least as this cast of the interior of the skull is capable of demonstrating.

Swift of course was not exempt from intercurrent diseases, the most important of which appear to have been a severe colic in 1696, which brought him to extremity, "so that all

despaired of my life, and the newspapers reported me dead;" an attack of herpes zoster of the left neck and shoulder, with its atrocious after-pains, in 1712; and a severe attack of ague in 1720, which lasted a whole year. But these maladies appear to have passed away without permanent injury, and it is to the cold which, before he was twenty, gave him a deafness and an ear "which has never been well since," to which we must look as the origin of his physical and ultimately of his mental miseries, and which made him often describe himself as

"Vertiginosus, inops, surdus, male gratus amicis."

He does not appear ever to have guessed that his vertigo was due to the state of the auditory organ, nor indeed did any physician ever suspect that such was the case in any one suffering from ear-giddiness, until, led to reflect on the subject by Fleuren's experiments on the semicircular canals of pigeons and rabbits, Ménière recognised the causal connection in 1861. The paroxysmal nature of the affection, with long intervals of immunity, is well marked in Swift's case, although it would appear from a quotation made by Forster, p. 253, from one of his note-books, that the fits were pretty frequent, the reference to fits in his journal and correspondence only applying to the more serious occasions. The quotation is as follows:

"1708. *Nov.*—From 6th to 16, often giddy. G^{d} help me. So to 25 less. 16, Brandy for giddiness 2*s*. Br^{dy} 3^{d}. *Dec.* 5.—Horribly sick. 12*th.*—Much better, thank God and M. D.'s prayers. 16*th.*—Bad fit at Mrs. Bartons. 24. Better; but dread a fit. Better still to the end. 1709, *Jan.* 21*st.*—An ill fit but not to excess. 29. Out of order. 31. Not well at times. *Feb.* 7.—Small fit abroad. Pretty well to the end. *March.*—Headache frequent. *April* 2*nd.*—Small giddy fit and swimming in the head. M. D. and God help me. *August.*—Sick with giddiness much. 1710. *Jan.*—Giddy. *March.*—Sadly for a day. 4*th.* Giddy from 4th. 14*th.*—Very ill. *July.*—Terrible fit. G^{d} knows what may be the event. Better towards the end."

It is true that the paroxysms were not so numerous as those of most other cases of the disease which are on record, and also that the deafness never became absolute, and therefore

that the disease never ceased. It increased in intensity as life advanced, until it confined him to his chamber for weeks at a time. It is also to be remarked that a slight degree of vertigo caused great constitutional disturbance. "I had a little turn in my head this morning, which, though it did not last above a moment, yet, being of the true sort, has made me as weak as a dog all this day."—*Journal*, Oct. 23, 1711. "This morning I felt a little twitch of giddiness, which has disordered and weakened me with its ugly remains all this day."—*Journal*, Jan. 25, 1812. Another characteristic of the vertigo, noted in a quotation already given, is that at one time any slight movement of the head brought it on.

It is certain that this fearful disease, aggravated with the increase of years, had an influence in the causation of Swift's insanity; but that its influence was direct, that is to say by the extension of the local disease to the brain, is by no means so sure as its indirect effect as one source of the profound depression which marked the latter years of his sane life. We have no authentic account of the first outbreak of insanity, and Sir Walter's statement that, after his understanding failed, "his first state was that of violent and furious lunacy," would seem to have been applicable only to that later period when he suffered indescribable torture from some unknown local disease, producing exophthalmos of the left eye. It is clear there was emotional depression amounting to melancholia, and "much water in the brain," which was probably sub-acrachnoid effusion, is sufficient evidence of dementia. But there was also that form of aphasia in which scraps of reasonable language come automatically, though intentional effort can produce no words, and very curiously in connection with this fact comes the evidence of the plaster-cast, "brought to light a hundred years after death," that there was right-sided hemiplegia. The knowledge of the importance of this fact also has been acquired since Sir William Wilde wrote his work, and it is not therefore surprising that while he so carefully and skilfully marshals the data upon which our diagnosis is now made, he does not connect the right-sided hemiplegia with the very peculiar affection of speech recorded by one of the two authentic witnesses above quoted. Sir William Wilde ex-

presses the opinion that the hemiplegia had existed for several years before death, "for we find the same appearance much glossed over by the artist, together with a greater fullness or plumpness of the right cheek shown in a very admirable bust, probably the last ever taken." But as Wilde admits, the "six well-known busts of Swift, undoubtedly taken about the same time, exhibit six different forms of head bearing but little resemblance to each other," the much glossed over appearance can therefore scarcely be admitted as evidence. Probably the stroke of palsy recorded in the plaster cast had taken place unobserved at or about the time of the actual outbreak of the mental disorder, which might have masked the physical symptoms from observation.

When "the Vandal desecration of monuments" in 1835 exposed Swift's skull to the phrenologists, the great Dublin aurist might possibly have found in the bones of the ear traces of the cause of his giddiness. When Mr. Whiteway examined the brain, he might have found the cause of Swift's right-sided hemiplegia and his aphasia. It is enough now that we can diagnose his lifelong disease as labyrinthine vertigo, and his insanity as dementia with aphasia; the dementia arising from general decay of the brain from age and disease, the paralysis and aphasia from disease of one particular part of the brain.

With all the tortures of the lifelong disease from which he suffered, and its obvious effect upon his temper in his later years, it is wonderful that Swift did retain his reason until, in the seventy-fourth year of his age, he was in all probability struck down by a new disease in the form of a localised left-side apoplexy or cerebral softening, which determined the symptoms of his insanity.

That Swift's works contain no indications of insanity appears to me certain. As well say that Shakespeare was mad because he wrote a good deal which we think nasty. In the fashion of the day, Swift was too prone to make what may be called excrementitious jokes and jibes. But that perfect gentleman Antonio voided his rheum upon Shylock's beard; and the same kind of thing runs through our literature, no one objecting, until we rather recently began to become less natural and more

nice. Some of our smaller humourists and men of letters have criticised this great king of humour as if he were both bad and mad, not perceiving that if he were really insane he must be pitied and not cursed. But it is the weakest of arguments to say, with Festus, for want of argument, "much learning doth make thee mad." There is always weakness in madness, but there is little sign of this in Swift's works. There is always some inconsequentness or incoherency in madness, but there is none of this in Swift. Down to that last letter to Mrs. Whiteway he is most wretched, but he is still collected and wholly himself.

One final consideration is that the oppressive and disabling nature of Swift's lifelong disease has been greatly underrated in the more severe of the criticisms which have been made with regard to his conduct to Esther Johnstone. I do not know that labyrinthine vertigo would necessarily incapacitate a man for the performance of marital duties, but it certainly might be a barrier to them more formidable than unprofessional critics are likely to suppose possible. Dr. Beddoes suggested that Swift was impotent from youthful dissipation, of which there is not a tittle of evidence. May not the great and grave disease of which I have adduced such copious evidence have been the real reason why Swift did not live with the woman whom it is certain that he loved with the most tender and persistent devotion?

Critical Digests and Notices of Books.

Etudes Cliniques sur l'Hystéro-épilepsie, ou grande hystérie. Par le Dr. PAUL RICHER. Paris, Delahaye, 1881: xvi.-734 pp.

THE activity displayed in the study of hystero-epilepsy and the curious phenomena allied to it, has resulted in the formation of a large literature, in the shape of innumerable papers and articles dispersed in numerous periodicals. The interest taken in the subject goes on increasing every day, and Dr. Richer deserves our best thanks for having undertaken the task of reducing the multitude of loose material to an orderly though comprehensive digest. Our aim here will be less to criticise than give a condensed account of the work before us.

The typical hystero-epileptic fit is usually heralded by a period of prodromal symptoms, affecting one or more functions. Thus flatulence, spasm of the throat, ptyalism, dyspnœa, palpitation, vasomotor disturbances, and other similar signs may be present in the sphere of organic life. The muscular system may be the seat of various symptoms, such as loss of power, tremors, transitory or persistent contractions. In the sensory sphere, anæsthesia, complete or partial (frequently affecting one-half of the body), and involving the special senses as well as the common cutaneous sensibility. Painful symptoms are not rare, constituting the aura. The latter, as Charcot has shown, stands in intimate connection with the ovarian tenderness, which is pretty uniformly present at all times in hystero-epileptics. Pressure upon the tender spots calls forth a series of very peculiar sensations, which follow a definite course, clustering at first about the ovary, next about the throat, and finally in the brain, and are the same as those observed in the spontaneous aura. Many patients present other points on the

body besides the ovarian region, pressure on which is followed by the aura and the fit. These have an obvious resemblance to the epileptogenous zones of Brown-Séquard's guinea-pigs, and hence have been called hysterogenous zones. These zones are very variously distributed, and not nearly so frequently confined to the hemianæsthetic side as the ovarian tenderness is. It may be added here, that pressure on all these points are frequently as effectual in cutting short the fit—by substitution or interference, as it were—as in determining it. Lastly, among the prodromal phenomena, we must mention psychical disturbances. The moral and intellectual processes are variously distorted, and hallucinations play an important part in the picture. These take sometimes an erotic turn of remarkable violence, leaving the patient during her conscious hours under a vivid impression of their reality; but more frequently consist in visions of animals and the like, which, curiously enough, persistently make their appearance on the hemianæsthetic side.

It has been the merit of Prof. Charcot to seize, among the apparently incoherent, proteiform phenomena of the hystero-epileptic attack, upon a fundamental order of sequence, to which they can usually be reduced. His generalisations have been amply confirmed by the subsequent observation of new cases either at the Salpêtrière itself, or in other hospitals both in France and abroad, as well as by the careful study of records made by competent observers of earlier times. The records of the demoniac possessions which played such an important *rôle* during the Middle Ages, yield many unexpected points of resemblance between the phenomena occurring under so very dissimilar circumstances, and described in so very different a spirit, and those which form the subject-matter of the volume before us.

The first period of the hystero-epileptic fit is the only one which bears any resemblance to the true epileptic seizure; but, it must be confessed, this resemblance is very close indeed. The prodromal phenomena culminate into loss of consciousness, arrest of respiration, and muscular rigidity. We cannot enter here into any details of the phenomena observed, but must refer the reader once for all to the beautiful illustrations

and the myographic tracings with which Dr. Richer has enriched his book with no stinted hand. Let it suffice for us to say here that the epileptoid period consists of several phases characterised—(1) by tonic contractions with large, slow, partial movements, and presently by universal tetanic immobilisation; (2) by chronic contractions; (3) by resolution. All this, as we said, is very like true epilepsy, but is differentiated from it by the absence of the peculiar cry often uttered by the patient in the latter disease at the beginning of the fit, and by the possibility of bringing the patient to consciousness by ovarian pressure, or voltaic alternatives of a galvanic current.

The second period of the fit succeeds closely to the first, and is aptly designated by Charcot under the name of Clownism. The patient is no longer unconscious, as proved by the fact that if brought round by ovarian compression she is able to recollect what was passing through her mind—though still insensible to the usual external stimuli; she passes through a phase of contortions and a phase of *grands mouvements*. During the first, a tonic spasm of considerable duration throws the body into a series of attitudes defined as "illogical" by Charcot (from their non-correspondence to any emotional or intellectual states). A common form of these attitudes is the *arc de cercle*, or one of its derivatives. When fully developed, the latter consists in a complete opisthotonos, the patient resting upon her toes and forehead. During the phase of the *grands mouvements* there occurs, after a piercing cry has been uttered once or oftener, first a series of more or less rhythmical movements of great amplitude (the legs and trunk, for instance, being thrown high up in the air, or the head and trunk "*saluting*"), and next a scene of the utmost fury, during which the patient yells like a wild beast and struggles wildly, tearing up everything, striking out, and twisting her body in all directions. This part of the attack is the one which may assume the characters of a true demoniacal possession.

We now come to the third period, that of the *passionelles* or emotional attitudes. Here the patient's mind is the prey of hallucinations, which she acts outwardly by throwing herself into the appropriate postures. She is completely insensible

to external stimuli, but when brought out of her fit can still remember what was passing through her mind at the time, and give an account of it which fully tallies with the external manifestations observed. During this period there is frequently a copious flow of words, which give a clue to the different scenes through which her hallucinations carry her. A great proportion of these usually take their origin in memories of her past existence. Events she has witnessed, persons she has known are mixed up with visions of purely imaginary objects, and every passion of the human mind finds its delineation in the remarkable display of "poses plastiques" through which the patients goes during her delirium. Religious ecstasy, joy, anger, spite, sorrow, love in all its phases, from the purest affection down to the most cynical lubricity, with a hundred transitional forms, and every possible combination, all are embodied with the art of the most consummate actor. The flow of thought is often remarkably rapid, and the passage from one mood into its opposite sometimes sudden in the extreme.

The transition between the period of emotional attitudes and that of waking consciousness is bridged over by the fourth period, that of delirium. Here the patient acts less and talks more. Usually the memories of her past life furnish the themes of long soliloquies; but the fact that the consciousness of external things is returning is proved by the illusions to which the patient becomes subject: sights and sounds are perceived, but perverted into factors of her morbid train of thought. Hence the delirium of the fourth period is far more diversified than that of the third, which in many cases is stereotyped, so to speak, the same events here ever recurring in the same order in the mind of the patient, and giving rise to the same display of embodiments of psychical states.

The delirium of the last period, however, is not always uniform and quiet; occasionally hallucinations of variable nature trouble the patient. The visions are sometimes placid, sometimes horrible; the latter are chiefly of animals (zoöpsia). Another feature which often marks the end of the fit is the occurrences of generalised or partial muscular spasms. The patient's body may be twisted into every conceivable shape.

But the following differences may be noted between these and the truly epileptoid contractions; first, that the muscles of respiration are here unaffected; second, that consciousness is present, and the patient is suffering intense pain. On the other hand, old standing hysterical contractures (and paralysis) have been known to disappear at this stage of the fit, which, we may add, may be protracted over some hours or even days.

The typical hystero-epileptic fit, as just described, with its four periods distinctly characterised, is not often met with. It is subject to many variations. For instance, patients often instead of having a single seizure, fall into an *état de mal*, in which numerous fits follow one another, not by mere succession, but by imbrication as it were; that is, the next fit breaks out before the previous one has run through the complete cycle of phenomena. In other cases one (or more) period is developed at the expense of the others, which may be absent, or at least but very transient and faintly accentuated. Others again present phenomena due to the interference of lethargic, somnambulistic, or cataleptic accidents, more or less developed in the course of evolution of the fit.

The first or epileptoid period may predominate, for instance. The patient then may fall into an epileptoid *état de mal*, lasting days or weeks, during which the temperature remains nearly normal (in true epilepsy this condition is marked by a considerable rise). Or she may present a series of incomplete epileptoid fits, similar to a true epileptic vertigo, with a few localised muscular contractions. When the contortions of the second period are carried to their utmost pitch, and accompanied by more than the usual manifestations of the wild and blind fury which we have seen mentioned above, a true "demoniacal possession" is witnessed. Or again, the fearful character implied in this term may be wanting, and mere "clownism," but pushed to its highest degree of development, is observed. The display of muscular power and violence in such cases is perfectly astonishing on the part of otherwise harmless, and it may be delicate, women.

The third period may represent wholly, or nearly so, the hystero-epileptic fit; and then we have in some cases the spectacle of a religious ectasis, in which the patient goes

through the performance of a whole series of acts connected with her religious belief and emotions. The Roman Catholic hagiography is rich in accounts of such cases; and no doubt those of the modern saintly women alleged to be the object of special divine favours, and to prove their claims by exhibitions of this kind, who happened not to be impostors, were simple hystero-epileptics.

Under the name of acute hysterical madness, Griesinger describes the fourth period, that of delirium, when it forms the bulk of the hystero-epileptic fit. It is to be noted that though at times some spasmodic or other factor of the fit accompanies hysterical delirium, the latter may supervene quite independently. Dr. Richer mentions the following points with reference to it:—Conscientiousness of the delirium; analogy with tonic delirium and with somnambulism; influence of present preoccupations and past emotions; exaltation of the intellect; nobility of the ideas; perversion of thought and feeling; fixed ideas; automatism; irresistible impulse; simulation; agitation and stupor. We have spoken above of the frequency of erotic ideas and of hallucinations and illusions.

The readers of 'BRAIN' will find in Vol. I., p. 558, a short account of the first experiments made by Prof. Charcot on hypnotism and the allied conditions, in hystero-epileptics. Dr. Richer gives us a complete summary of all that has been done at the Salpêtrière in this direction. Those who have had the advantage of witnessing the phenomena themselves will agree that they are of the highest promise towards the eventual elucidation of many physical problems, and are an irrefragable proof of the very small part played by "expectant attention" in the once much-abused performances of hystero-epileptics at the Salpêtrière.

The phenomena observed may be grouped under four headings: 1. Cataleptical state; 2. State of suggestion, or automatism; 3. State of lethargy, with muscular hyperexcitability; 4. The same without hyperexcitability, or somnambulism. The author fully illustrates these conditions, and explains the various modes of production and characteristic manifestations of each. We can here give but a succinct account of the facts observed, and commend Dr. Richer's pages to the attention of

the reader interested in the question. Many hysterical patients are thrown, by fixing their eyes upon a bright light, into a state of catalepsy similar to the idiopathic condition so named. There is absolute anæsthesia, waxy flexibility of the limbs, and the curious adaptation of the facial expression to the attitudes given to the limbs. Thus, prayer or threat, love or disgust, may be successively depicted on the countenance when the arms are placed in the corresponding positions. Closure of the eyelids, or sudden removal of the light, converts the cataleptical into a lethargical condition (somnambulism, magnetic sleep, &c.). Here complete muscular resolution prevails, often accompanied with muscular hyperexcitability to the weakest stimuli. Touching or rubbing the skin at any point throws the underlying muscle into a state of contraction, exactly as localised faradisation would. Each of the small muscles of the ear, face, or hand, for instance, may be singled out; or if the facial or ulnar nerve be irritated by pressure, all the muscles innervated are thrown into violent contraction. This contraction may persist for a very considerable time (in the case of the trunk and limbs, not the face) after the patient has been awakened, and presents the same phenomena of transfer as the idiopathic hysterical contractions. During this lethargical condition, clonus of the upper eyelid and of the eyeball-muscles is observed. The anæsthesia is complete. Then follows a condition more allied to somnambulism, in which the patient may be made to perform various acts to order. To awaken the patient it is enough to blow in her face, or press over the ovarian region. The relationship between the lethargical condition and the true convulsive attack is shown by the epileptoid phenomena which accompany its beginning and its end: pharyngeal spasm, foaming at the mouth, &c.

The same patient may be made hemicataleptic and hemilethargic at once by closing one of her eyes when cataleptised in the usual way. The side of the body on which the eye is closed becomes lethargic, whilst the other remains cataleptic.

The contractures produced during the sleep may be resolved by stimulation of the opponent muscles, or by awaking the

patient. They are fixed by bringing her back into the cataleptical condition before awaking her.

Other agents, besides light, cataleptise hysterical patients; thus, the vibrations of a tuning-fork fixed on a resonator, and a sudden loud sound are very effectual. Cases are recorded where patients were seized on hearing the cymbals suddenly used in an orchestra.

Pressure upon the eyeballs, of the fixing of any near object by the patient, produces the lethargic condition, which is readily converted into the cataleptical by merely raising the eyelids; whilst pressure on the vertex gives rise in most hystero-epileptics to a variety of the lethargical condition characterised by the absence of muscular hyperexcitability, and the impossibility of converting it into catalepsy by raising the eyelids. The patient also presents certain psychical phenomena more allied to true somnambulism. The cataleptical condition may be after a while readily made to pass into a state of automatism, in which, though unconscious, the patient may be made to perform series of actions connected with impressions produced on her senses. Thus she can be made to reproduce, with the accuracy of the image in the looking-glass, the movements of the experimenter placed in front of her, or carry out series of movements suggested to her by placing her limbs in appropriate attitudes, such as climbing up a ladder, creeping on all-fours, and the like. Or again, if an object of known use be placed in her hands, she immediately sets herself to carry out the train of actions suggested by it. It is easy also to call forth hallucinations in her mind by verbal suggestions; whispering in her ear, "Here are flowers," "There comes a serpent," and the like, suggests whole trains of thought which give rise to appropriate actions and language, frequently of the most lively description.

It is to be remembered that all patients are not susceptible of being roused out of the cataleptical condition, and that whilst in the lethargic state some may also be made to display the phenomena of automatism. The lethargy without muscular hyperexcitability presents also some curious phenomena upon which the author wisely abstains to dwell, because they are as yet but little explored, and might readily be turned to account

by charlatans. Thus the patient may be made to answer questions, not only with sense, but sometimes far more intelligently than in her waking condition. She performs acts with the greatest accuracy, and is readily thrown into subjection to one individual, who then alone has the power of starting the automatic actions. Nay, each half of the body may be so subjected, independently, to two experimenters.

We cannot enter into any details with reference to the varieties of the hystero-epileptic attack due to the idiopathic immixtion of lethargic, cataleptic, and somnambulistic phenomena; nor upon the analogies between the hysterical delirium and the cerebral effects of certain drugs, such as hachisch, alcohol, absinthe, opium, &c. The reader will find these topics fully illustrated and discussed in Dr. Richer's work. In the third part he also discusses various topics, which, being probably familiar to our readers (hysterical anæsthesia, contractures, &c.,) we pass over to reach the last chapters of the work concerning the diagnosis and treatment of hystero-epilepsy. Much has been said about the influence of simulation and expectant attention in the causation of the phenomena attending hysterical manifestations. Several writers in England, and elsewhere, who, by the way, did not always take the trouble of going over to Paris to investigate personally the question, have assumed that Prof. Charcot and his disciples were entirely ignorant of the sources of fallacy attending their researches. Dr. Richer's volume will do much to dispel any such impression that may yet subsist. The results of numerous independent tests that have been applied to the phenomena, and the uniformity displayed in the order of their manifestation, are amply sufficient to carry conviction into any unprejudiced mind.

The following points are pathognomonic in distinguishing true epileptic from hystero-epileptic attacks. The latter may be arrested by pressure over the ovarian and other regions of the body, and by electrical shocks (voltaic alternatives), but are not influenced by the bromides. The temperature remains about normal in the hystero-epileptic *état de mal;* and patients suffering from this disorder do not, even after years, present any of the psychical disturbances so common among the true epileptics.

The therapeutics of hystero-epilepsy fall under two heads: 1st, management of the attacks; 2nd, treatment of the patients between the attacks. The former consists in cutting short the fits, or diminishing their frequency, by such measures as compression of the ovaries with the hand or tourniquet, electrical currents, and inhalations of chloroform, nitrate of amyl and the like. The latter, more successful than might be supposed, considering the fearful manifestations of the disease, consists chiefly in the removal of the patient from her often unsatisfactory surroundings and, in addition to moral influences, hydrotherapic and electric applications. The latter are chiefly of the static order, and Dr. Vigouroux (who has charge of this department at the Salpêtrière) has obtained some very interesting results. The static bath (or charging of the patient on an insulated stool) has very marked effects on the hemianæsthesia, for instance, and produces immediately the phenomenon of transfer with consecutive oscillations.

The historical appendix which concludes Dr. Richer's book is by no means the least interesting part of it. He intends to show that hystero-epilepsy is not a new disease, as might be thought from the silence of the earlier medical observers, but that, under various names, forms, and circumstances, it has existed from early times. Striking analogies will be found, for instance, between the descriptions given of the epidemic chorea of the middle ages (14th century) and those of the present hystero-epileptic phenomena. Again, the demoniacal possessions, of which we have witnessed examples within the last few years. The singular epidemic which broke out in Paris 150 years ago, that of the "Convulsionnaires de Saint-Médard," has been carefully described by competent observers, and offers many typical instances of the disease as now observed at the Salpêtrière. In Protestant countries such phenomena are of course unfrequent; still, the scenes witnessed at American "camp meetings," at Irish "revivals"[1] have been so frequently characterised by peculiar sensory and motor disturbances on the part of those who took part in them, that we cannot ignore the neurosis which lay at the root of such manifestations. Again, the ecstatic epidemic of Sweden (1841),

[1] See 'Christian Revivals; their History and Natural History.' By J. Chapman, M.D., M.R.C.P. London, 1880.

and the lives of "holy" women from Douceline (1274) to Louise Lateau (1868), furnish the author with many points of comparison between the so-called religious and pathological phenomena he has so well described.

Dr. Richer is a first-class artist, and his work is made far more instructive, as well as more attractive, by the numerous plates and illustrations which so faithfully reproduce the various phases of the hystero-epileptic fit in all its varieties. The volume before us is an able, complete, and trustworthy account of all that is known of the subject it professes to treat.

A. DE WATTEVILLE.

Kundrat on Porencephalie. (Die Porencephalie eine Anatomische Studie. Von Dr. HANNS KUNDRAT. Graz, 1882. 4to.)

THIS work is a monograph on the morbid condition of the brain, to which the name Porencephalie was first given by Heschl. It is characterised by the occurrence, mostly on the surface of the cerebral hemispheres, of hollows due to loss of substance, either opening into the arachnoid cavity, or separated from this by the visceral arachnoid, extending to various depths into the medullary substance, reaching the ependyma, or even penetrating completely into the ventricle. These defects were for a long time considered of a congenital nature. Yet this is not universally so, nor is their origin thereby explained. To this point our author specially directs his attention. He has been fortunate enough to obtain as many as twelve original cases of his own, and as well as to examine the collection made by his predecessor Heschl. Besides these, reference is made to cases reported by Cruveilhier and others with sufficient precision or illustration as to render them available for his purposes.

In all he deals with forty-one cases.

He finds that porencephalie is an anatomical defect produced by destructive lesions, which in their origin and nature do not differ from those caused by hæmorrhage, thrombosis, embolism or anæmia. This peculiarity consists only in their

seat on the surface, and in their form, which depends on the completeness of the destructive process. These defects may occur *in utero*, and are therefore congenital, or they may be developed at any period of extrauterine life. The former are by far the more frequent and typical. They affect the cerebral cortex, and are conditioned mainly by anæmia of certain cortical arteries, most commonly of the middle cerebral. When hydrocephalus is also present, the affection may extend over the whole cerebral mantle as far as the basal ganglia. When congenital, these defects injuriously affect the development of the brain. They cause changes in the direction of the convolutions, the convolutions disposing themselves radially to the centre of the hollow. They also impair the development of the individual, and lead generally to idiocy, and frequently to inability to use articulate speech.

If the motor regions of the cortex are affected, paralysis with contracture is always produced.

The cases which occur in extra-uterine life, much less common than the preceding, are mostly caused by embolism or hæmorrhage, but may be the result also of injuries. These do not disturb the general form of the brain and the disposition of the convolutions, but when they occur at an early age they prevent normal development and lead to idiocy. In these cases there may be aphasia, but not absolute abolition of the speech faculty. Paralysis with contracture occurs also in these cases when the motor zone of the cortex is the seat of lesion. A similar condition results also from lesion of the ganglia and internal capsule. In the latter case descending degeneration ensues.

The congenital and acquired defects may also lead to symmetrical or asymmetrical changes in the skull, either of the nature of enlargement or the reverse. When only one side of the skull is misshapen, it is mostly collateral with the cerebral defect.

The porous defects of the cerebral cortex may become cicatrised and closed, but their effects nevertheless continue.

These are only the leading points of a valuable monograph carefully prepared and illustrated.

DAVID FERRIER.

Exner on Cerebral Localisation. (Localisation der Functionen in der Grosshirnrinde des Menschen. Von Prof. SIGMUND EXNER. Wien, 1881.)

THE conclusions which Prof. Exner has arrived at from an analysis of the facts of disease as set forth in this work have already been noticed ('BRAIN' XI., p. 416). The methods which he has followed are here described in detail and copiously illustrated. Many reasons might be stated why the conclusions which Professor Exner has come to cannot be accepted as an accurate representation of the teaching of the facts of cerebral disease. His statistics of cortical lesions are vitiated by the inclusion of a large number of tumours, the symptoms in connection with which are obviously due to the indirect action on other parts than the immediate seat of the disease; and his doctrine of relative centres, founded only on the *frequency* with which lesions of these regions cause certain symptoms, cannot be regarded as based on conclusive evidence.

DAVID FERRIER.

A Treatise on the Diseases of the Nervous System. By JAMES ROSS, M.D. Two vols. J. and A. Churchill.

This is a work of very great value. It is very well written, and is as simple as the very complex nature of the subject admits of. Dr. Ross does not artificially simplify his subject by the lazy process of ignoring its difficulties. He considers it clinically, physiologically, pathologically, and does not fail to take into account the psychical side of what is physically nervous action. He recognises the great value of Herbert Spencer's work as bringing into fundamental harmony many superficially different phenomena of nervous diseases. There is no other work on Diseases of the Nervous System which covers so much ground, and is yet so harmonious in its several departments as is that of Dr. Ross. It is very difficult for a reviewer to do the author justice. It is too good a work for mere praise. We can only remark on some sections of it.

Quotations will be given as samples of the author's acuteness, originality, and power.

The work is divided into two books; the first is on the general pathology of the Nervous System, the second on its special pathology. The first chapter of book 1, about seventy pages, is on the structure and functions of the Nervous System. This is not the usual dry compilation from most recent writings on anatomy and physiology, although it gives most recent knowledge. Besides much originality, in the usual acceptation of that term, there is displayed throughout the chapter the originality of the man who thinks for himself. The headings of some of the sections will suffice to indicate the enlargement Dr. Ross gives to his topic; "Antagonism betwixt the Size of Units and Absorption of Nourishment." He writes, vol. i, p. 13, "that an increase in the size of a cell is followed by a relative diminution of material exchanges is readily proved by the fact that the surface which a large cell presents for the absorption of nourishment, does not increase in a degree proportionate with its bulk. The mass of a body increases as the cube, while the surface only increases as the square, of the dimensions. When, for instance, a cell has doubled its dimensions, its mass is eight times, while its surface is only four times, the original size. It is evident, therefore, that a small cell presents, in proportion to its bulk, a larger surface to its environment for the absorption of nourishment than a large cell, and, consequently, material exchanges take place more readily in the former than in the latter." Other headings, are "Differentiation of Structure, and Specialization of Function," "Integration of Structure," "Passage from the general to the special in both structure and function." The whole chapter is a splendid contribution to Neurology, and should be minutely studied. We quote the last two sections of it, partly because they give a slight summary of some important facts in the earlier sections. There is another reason. Many persons suppose that evolution is concerned only with the evolution of animal life, with what is commonly called Darwinism. Students of Spencer's System of Philosophy know better. Dissolution is the word Spencer has long used for the process the opposite of evolution, and is, therefore,

rightly applied to the results of disease of the Nervous System.

"*The Law of Evolution.*—We have now passed very rapidly and very imperfectly under review the fundamental laws of nervous structure and function. We have seen that the nervous system consists essentially of cells and fibres, that the cells are first small, round, and uniform; that they gradually become large and assume numerous processes; that the fibres are at first small fibrils, which together form larger fibres; that these become complicated by assuming an elastic sheath, and still further complicated by assuming a second sheath of a very special character. We have seen how the fibres come to be packed together to form white cords, and the cells to form small masses of grey substance termed ganglia; how the cords integrate to form thick masses of white substance and the small ganglia to form masses of ganglionic grey substance, and how this continuous process assumed a still more complicated form when some of the ganglia became subordinate, while others exercised superordinate functions. The whole of the intricate processes here described illustrate the one great law of evolution. That law may be described as a progressive integration, both of structure and function, during which there is a passage from the uniform to the multiform, from the simple to the complex, and from the general to the special. During the evolution of the nervous system of man, the fundamental portion is first developed. The nervous system of man is at first similar to that possessed by all animals which possess a nervous system, or, at any rate, all those which are sufficiently elevated to possess a spinal cord; but as development proceeds, the nervous system of man becomes gradually differentiated from that of an ever-increasing number of the lower animals, while still maintaining a general likeness to the nervous system of the higher animals up to the time of birth. This, then, constitutes the *fundamental* portion of the nervous system of man; but after birth the *accessory* portion, which up till this time only appears in a rudimentary condition, now undergoes progressive development, and the nervous system of man becomes gradually differentiated from that of all other animals. It will thus be seen that the fundamental portion is first de-

veloped, and that the superaddition of the accessory portion greatly increases the multiformity, the complexity, and the speciality of the human nervous system, and it is, consequently, the latest product of its evolution."

"*Law of Dissolution.*—We must now proceed to regard the phenomena of the structure and function of the nervous system from a new and opposite standpoint. We must watch the cells lose their processes, and from the multiformity of the caudate cells with numerous processes pass to the uniformity of the round cells destitute of processes; we must observe the fibres losing their medullary sheath, then their elastic sheath, and finally the axis-cylinder itself becoming disorganised, so that the nervous tissue gradually gives place to a simple and uniform connective tissue; we must observe accompanying this process a corresponding loss of function in which the complex movements that characterise health become difficult or impossible; in one word, we must trace the records of a process in which the progressive integration, during which the phenomena of structure and function, instead of passing from the uniform to the multiform, from the simple to the complex, and from the general to the special, manifest a reverse tendency, of passing from the multiform to the uniform, from the complex to the simple, and from the special to the general. The law which governs this process is the law of dissolution, and it is the great law which regulates the phenomena of disease of the nervous system, just as evolution is the great law which regulates its growth and development."

It is scarcely necessary to add that the phenomena of evolution manifested in the growth and development of the organism are exceedingly gradual and continuous, and consequently the operation of the law of evolution can very readily be traced. Disease being, on the other hand, often sudden and violent in its onset, striking at times at the fundamental, at other times at the accessory portions of the nervous system, now producing its baneful influence at one stroke, again acting fitfully, and only on rare occasions, in a gradual and progressive manner; it may be inferred that the operation of the law of dissolution can never be so clearly

traced amongst morbid phenomena as that of evolution in the development of the organism. Nevertheless there are some diseases of the nervous system which are gradual in their invasion and progressive in their course, and in them the operation of the law is clearly visible; and it is astonishing how glimpses of the law may be obtained, even when the disease is sudden in its onset and rapid in its progress. One important corollary may be drawn from what has been said; that as the *accessory* portion of the nervous system is the last to be developed, it is the portion which is most liable to become diseased. Several reasons might be given why this should be the case, but these will appear in the subsequent pages. It will suffice at present to say that the accessory portion, from the late period of its development, is less stable than the fundamental portion, and that its necessarily frail structure will render it more liable to suffer both from accident and the inroads of disease."

The first part of Book II. contains eight chapters dealing with affections of peripheral nerves, cranial and spinal. It is a very clear account; the difficult things are rendered simpler by help of many illustrations. In the case of motor nerves, both spasmodic and paralytic affections are considered. The next part is on diseases of the sympathetic system, under which Dr. Ross gives a very elaborate account of headaches. The description of migraine is very excellent. An account of exophthalmic goitre is given in this Part.

Diseases of the spinal cord are then considered; to the account of them we can give the highest praise. But the next Part, "Diseases of the Encephalon" is perhaps the most remarkable in the work. The "Anatomical and Physiological Introduction" shows the author's method well, and illustrates his grasp of neurology. It, like other parts of the work, contains many illustrations, and by them and the author's lucid exposition, a very complex subject is made comparatively easy. A careful account of the topography of the cerebrum and cerebellum is given, and of the plan of arterial supply of the different divisions and sub-divisions of the encephalon. Practical ends are never lost sight of; relations of the convolution to parts of the skull are stated and illustrated; orderly "dissections," if we

may use the term, of the brain are made, so that at post-mortem examinations exactness in recording the locality of lesions is rendered easy. The minute anatomy of the brain is dealt with; the researches of Lockhart Clarke, Meynert, Bevan, Lewis, and others being made use of. A brief account of some of Herbert Spencer's views on cognitions and feelings is given under the head of 'Functions of the Cortex of the Cerebrum.' The following, from Section 686—*Anatomical Substrata of Consciousness*, is very interesting, and, we believe, very important:

"It cannot be supposed that the large cells, with the distinct processes and definite connections found in the internal division of the third layer of the cortex, will readily undergo structural changes in the healthy adult, and it is much more probable that any new alteration of structure in the cortex will proceed from the small cells of the external layers of the cortex. The first layer may probably be regarded as an embryonic layer without any active nerve functions, and consequently the second layer and external portion of the third layer of the cortex, the cells of which do not possess definite connections with one another, or with nerve fibres, must be regarded as the areas excitation of which is attended by the highest consciousness."

J. Hughlings-Jackson.

Clinical Cases.

A CASE OF PAROXYSMAL CLONIC SPASM OF LEFT RECTUS ABDOMINIS, WITH SYMPTOMS POINTING TO THE EXISTENCE OF GROSS INTRACRANIAL DISEASE.

BY E. BUCHANAN BAXTER, M.D., F.R.C.P.

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THE following case is put on record for two reasons. First, because it presents a somewhat unusual combination of symptoms, one of which—rhythmic spasm of an abdominal rectus—I have never before witnessed or seen described by others. Secondly, because of the unexpected failure of post-mortem inspection to throw any light upon the nature and origin of the disease. So much has been accomplished of late years to increase the certainty and precision of our diagnosis in intracranial disease, so many cases have been recorded in which a conscientious study of symptoms has enabled a correct opinion as to the nature and situation of morbid changes to be formed, that we are naturally prone to overestimate our powers in this direction. In the present instance, though unable to arrive at any very definite judgment as to details, I had no sort of doubt that I should discover some gross lesion after death.

Mrs. D., aged 35, first consulted me on November 18, 1879, for a nasal discharge, associated with peculiar "nervous attacks."

She was married in April 1877, and was then in perfect health. In September of the same year, after a good deal of worry and anxiety, she began to suffer from certain nervous symptoms, which have steadily increased in intensity and frequency, without undergoing any alteration in their character. They are of two kinds: headaches, and what she calls "twitchings." Shortly after these symptoms made their appearance, a clear watery fluid, sometimes rather offensive,

occasionally tinged with blood, began to come away from the right nostril.

She is a small, fairly plump woman, with rosy cheeks and no appearance of ill-health. Her manner is quiet and sensible, the reverse of "hysterical;" intelligence above the average; makes no attempt to excite sympathy or to exaggerate her sufferings. Menses regular. Has never been pregnant.

On closer enquiry, the following points were ascertained:—

1. *Headaches.*—These occur almost daily. The pain is most severe, beginning at the root of the nose, and spreading round to the back of the head. Moreover, there is a constant fixed pain, like clavus, referred to a point on the left side of the occiput, just outside the insertion of the muscles of the neck. On deep pressure with the finger-tip, a small area of tenderness can be made out here. The headaches are not at all periodic; they are always worse during the daytime, and never keep her awake at night. She distinguishes between these constant headaches and what she calls "bilious attacks." The latter have recurred every fortnight or three weeks during the past twelve months. They consist of violent headache, aggravated by light, noise, and movement, associated with nausea and eructation of a bitter fluid. She never vomits at other times. She suffers a good deal from constipation, and the state of her teeth interferes with proper mastication.

2. *So-called "twitchings."*—Hardly a day passes without an attack, which may be brought on at any moment by the most trifling emotion, even by a knock at the door. She had one in my presence the first time I saw her, and I copy the description written down at the time. "The first thing one sees is a jerking of the left shoulder downwards and forwards, recurring at intervals of two or three seconds. The jerks are very sudden and forcible. They go on increasing in violence for several minutes, though, by straightening her arms, clenching her teeth, clasping her hands, and holding her breath, she evidently does her best to repress them. On stripping her, the jerking of the left shoulder, and indeed of the entire left half of the trunk, is seen to be due to a violent recurrent spasm of the left *rectus abdominis*. The spasm is extremely sudden and violent, quite like the spasmodic contraction of the diaphragm in hiccup. As it increases in frequency and intensity, the pectoral muscles become involved, and the left humerus is simultaneously jerked forward. No other muscles take part in the movement. At the height of the paroxysm, a patchy erythema with irregular, sharply-defined margins, makes its appearance on her forehead and chin, spreading gradually over the face and neck in a deep rosy blush. She then begins to shed tears and laugh just like a

patient in an ordinary hysterical attack. Even when she is at her worst, she makes obvious efforts to control herself, and answers questions sensibly. The paroxysm passes off as gradually as it came on." The duration of the paroxysm varies from ten minutes to a couple of hours. When it lasts for an unusually long time, she falls asleep from sheer exhaustion, but continues to twitch feebly even in her sleep. The fits of laughing and crying always occur in connection with the "twitchings."

3. *Discharge from right nostril.*—Of this she complains bitterly. On examining through the mouth, the upper jaw is seen to be crowded with decayed stumps, the gums round which are slightly ulcerated. The posterior wall of the pharynx is smeared with thin muco-purulent matter, which has found its way down from the naso-pharynx. The right nostril is pervious. No ulceration or disease of bone can be detected by examining with the rhinoscope or from the front. The discharge is only sometimes offensive. I never could perceive any unpleasant odour when I saw her.

There never has been any loss of consciousness or intellectual confusion. There is no failure of muscular power, no hyperæsthesia or anæsthesia anywhere. No spinal or ovarian tenderness. No impairment of taste or smell. When the condition of her eyesight was enquired into, she admitted that it had been failing for some three weeks. "Things look misty, and I seem often to see only one half at a time." On examination, the pupils were found equal and to react well to eyes. No squint. Incomplete but decided hemiopia in both light. Double optic neuritis.[1]

There is uniform soft hypertrophy of the thyroid body, first noticed two years ago, when the present illness began. Pulse 104, regular. Nothing amiss with heart, lungs or urine.

She has repeatedly sought medical advice during the last two years. Her symptoms have always been attributed to hysteria. No treatment, however, has had any influence, either on the twitching, or on the discharge from the nostril.

I was disposed, at the first glance, to agree with the view taken by those who had previously seen her. The absence of any failure of health and nutrition, the onset of the symptoms some months after marriage, their connection with mental worry, the almost invariable association of simply hysterical

[1] Shortly afterwards, I had an opportunity of showing her to Mr. Nettleship, who made the following note about her eyes: "$V \frac{R}{L}$ 7 Jäger. $\infty \frac{12}{50}$. Colour-perception normal. Upper and outer quadrant of each f. v. either foggy or quite a blank. Neuritis of moderate intensity in either eye."

phenomena with the paroxysms of twitching—anomalous and even unique in my experience as these undoubtedly were—all pointed in this direction. But the discovery of the intra-ocular mischief speedily put me on the right track. Close observation, moreover, showed that the hysterical symptoms did not really form a part of the convulsive attack, but were engrafted upon it. They were due, as I convinced myself by subsequent investigation, to the emotional break-down consequent on the utter failure of the patient's vehement efforts to check or control the twitching. Again, her manner, the absence of any menstrual derangement, of any spinal or ovarian hyperæsthesia, were against the hypothesis of the functional nature of her malady.

Admitting, then, that the mischief was of organic origin, what was its nature and its seat? As regards the first question, the possibility of syphilitic infection was thoroughly considered, and after most careful examination of the patient and of her husband, rejected. It may here be added, that the absolutely negative result of anti-syphilitic remedies administered for a considerable time and in adequate doses fully justified this conclusion. The most likely hypothesis out of a number that presented themselves appeared to be that of some chronic disease of the body of the sphenoid or the neighbouring part of the ethmoid bone, leading to inflammatory changes about the optic chiasma and adjoining parts of the brain. Chronic mischief in this locality might account for the headaches, the neuritis and peculiar hemiopia, as well as for the discharge from the nostril. No theory of localisation would account for the twitching of the rectus muscle.

This, of course, was mere speculation. Of one thing I felt sure, that the symptoms were due to some coarse organic mischief, and that the prognosis was accordingly unfavourable. Of these two convictions, the second alone, as the sequel shows, turned out to be correct.

Here is a brief abstract of the subsequent notes of the case:—

January 6th, 1880.—No change in symptoms, save that her eyesight is failing. Ophthalmoscopic appearances the same as before, except for a small dotted hæmorrhage on the outer edge of the right disk. The headaches and "bilious attacks" as frequent and severe as ever.

Feb. 3rd.—Has not vomited since her last visit. Symptoms unaltered, save that for about a week her nights have been restless; she falls asleep and is roused by frightful dreams. The mucopurulent discharge from the right nostril is abundant, and occasionally streaked with blood.

May 25th.—Has felt a little better, on the whole, during

the last three months. Still looks plump and ruddy. Suffers a good deal from inframammary pain shooting through to the angle of the left scapula, with aching of the left arm. This is clearly due to over-fatigue from the frequent and violent twitching.

June 15th.—Has had two attacks of vomiting since her last visit. One was extremely severe and lasted for fourteen hours. Eyesight failing. No material change in fundus oculi. For the first time, I find her looking rather pale and tremulous. She ascribes this to the agonising and almost continual headache.

August 4th.—Vomiting has been very unfrequent. Otherwise no material change. No signs of atrophy of disk as yet.

Sept. 28th.—Still looking rosy and well. Sleep disturbed for a fortnight. She sleeps for five minutes and wakes up under the impression that she has been asleep for hours. The headaches have often been attended with severe vomiting. Nasal discharge persists. So does the thyroid enlargement.

Oct. 27th.—In a very tremulous state. Her headaches have been aggravated, during the last eight days, by frightful neuralgic pain in the left eyeball. The conjunctiva of this eye is intensely injected over its outer half. Eyeball tender on pressure; no increase of tension. Pulse regular, 100. Other symptoms unaltered.

Nov. 25th.—Looks really pale and ill. Outer half of left eyeball still much injected. Can just make out 19 Jäger with either eye and with a limited portion of each retina. Decided photophobia. Olfactory sense continues normal. No change in ophthalmoscopic appearances. Has not vomited for four weeks, though the pain across the temples and behind the root of the nose is as bad as ever.

This was her last visit to me. She ceased to be able to leave her home, where she was under the care of Dr. Poole of Sidcup, to whose courtesy I am indebted for what I know of her subsequent progress and for the opportunity of examining her after death.

She appears to have become steadily thinner and weaker, and to have suffered constantly from pain in the head and vomiting. She did not take to her bed, however, till about a fortnight before her death, which took place on the 29th of January, 1881. Three days before she died she had general convulsions, which recurred frequently and terminated in coma. Her intellect remained unaffected to the last, although a sudden change was observed in her frame of mind. Whereas she had previously, amid all her sufferings, remained cheerful and resigned, about ten days before her death she became pro-

foundly melancholy, taking the most gloomy views of her religious condition.

A word as to treatment. It has already been stated that a full trial was given to the iodide of potassium in large doses, and to mercuric chloride. They produced no effect. She took, for the relief of her symptoms, bromide of potassium, occasionally chloral hydrate and hydrobromic acid. The only relief to her headaches was given by small blisters kept open with Ung. Sabinæ. The decayed stumps were all extracted from her upper jaw, and she employed a nasal douche of potassic permanganate. This prevented the offensive smell of which she complained, but which was never very marked.

At the post-mortem examination, made by Dr. Poole and myself, the head only was opened. The body was thin, but not emaciated. No signs of decomposition. The bones of the skull appeared to be more thick and dense in texture than usual, offering considerable resistance to the saw. The interior of the skull, the brain, and its membranes, were very carefully examined in a good light, and nothing in any way abnormal was discovered. With bone-nippers I made my way into the cavities of the sphenoid and ethmoid, without finding any evidence of disease. It ought, perhaps, to be added, that no appearance was anywhere met with calculated even to excite suspicion and call for more minute examination.

The negative result of the inspection was a surprise to me. The experience of others may, perhaps, contribute to the explanation of what remains to me inexplicable, and it is with this hope that I publish the case.

A CASE OF TUMOUR IN THE MEDULLA OBLONGATA AND PONS VAROLII, WITH REMARKABLE PARALYTIC SYMPTOMS.

BY J. M. HOBSON, M.D., EDIN.

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[This report is only a fragment of what it should have been, but I trust that its intrinsic interest will justify its publication.]

THE case was admitted into the North-Eastern Hospital for Children in Hackney, on February the 27th, 1880, under the care of Dr. Sansom, with whose kind permission I now publish it from the notes which I made at the time.

Benjamin L., aged 2 years, was quite well and able to run about and talk till *nine weeks before admission*. At that time he began to waste, and to have difficulty in swallowing. *Five weeks* later he began to have some impairment—I was told by his father—in the movements of the left arm, and the left upper eyelid began to droop. Then this side of the body was said to get better, and the corresponding parts on the right side became affected in the same manner. But previous to the affection of the arms the legs were effected. The date of this, however, is uncertain, as the fact was obtained more than a year after the child's death.

On admission the child was certainly a most remarkable object. His body was very thin and his head very large, his face expressionless, and saliva dribbled from the corners of his mouth. He had no power of articulation, considerable difficulty in swallowing, and frequently made a noise in his throat, as if some saliva were there which could not be got rid of. The right eyelid *at times* drooped, but not constantly, and further observation showed that this was due to contraction of the orbicular muscle. There was no apparent squinting or inequality of the pupils. From time to time there were clonic contractions of the right side of the mouth. Not only was he incapable of standing when supported, but he was unable to sit upright in bed, and, when unsupported

and not lying back, his body was bent forward. The position of his head varied; sometimes it was drawn much back and towards the right shoulder. While the right arm was lying unused it was quiet, but any attempt at movement excited a *rhythmical jactitation* of the limb. At some of these times the whole body was jerked.

The optic discs were normal. The temperature was normal. The bowels were costive, but everything was passed under him.

On March the 5th I made the following note:—"It is clear that the left side of the face is paralysed. Saliva dribbles from either side. The movement of the head [above mentioned] can be induced by creating a necessity for its support by the child's own muscular effort [i.e. by removing all artificial support from the head]. Three times to-day, when the spasm was so evoked, the right sterno-mastoid became rigid and the chin was moved to the left; once the head approached the *right* shoulder, but on the other occasions this was not the case. There seems to be some tendency for the *left* arm to be jerked during voluntary movement. The rhythmical jerking of the *right* arm is very free."

On March the 9th I noted:—"The legs are sometimes drawn up stiffly. The left arm certainly jerks on voluntary movement. Once this evening the chin was moved to the *right* side, and at another time there was strong contraction of the right side of the face, and the head was drawn back."

On the night of March the 15th the patient was so ill that it seemed he could not survive till morning. However, on the 17th he was no worse, and could swallow better than he had been able to do for some time past. Both arms would jerk when he attempted to get hold of anything, and the fingers of the right hand were got round any object with difficulty. Ever since admission he had been very irritable, making a little cracked squealing noise when not pleased, and, at such times, only the right side of his face would be used.

He died on March the 22nd, a little over three weeks from admission. He had been getting lower for some time. When last observed, there was no obvious rigid drawing up of the legs. No marked changes were observed in the optic discs before death.

I have, unfortunately, no note at the time as to sensation. It would clearly have been impossible to gauge this with any precision.

Necropsy.—There was much subarachnoid œdema. The ventricles were filled with fluid to distension. No structural lesion was found in the brain till the medulla was inspected, which was *bulbous*, indeed—twice the normal size. On section,

a tumour was found occupying in an irregular manner the central and posterior part of the upper half of the medulla and, in the pons, being confined almost entirely to the left side (see Figs. 1–4).

Microscopically it was found that the growth was true tubercle, well showing, although much was caseating, the characteristic giant-cells with small round cells and trabecular framework (see Fig. 6).

Very unfortunately the sections were, in great measure, a failure, for, between their being cut and their being mounted, the majority fell to pieces, and the result is that I am not able to show a single good section of the medulla through the mass of the tumour. However, I have enough material to indicate in broad outlines the pathological anatomy of this case.

I give on the next page some drawings taken from the more successful specimens:

Fig. 1 shows the space occupied by the tumour at the middle of the pons. It reaches to the floor of the fourth ventricle, and will cut off, on the left side, all the vertical fibres, except a few anteriorly, and on the right side, a few fibres near the middle line.

In Fig. 2, a section through the highest planes of the medulla, none of the vertical fibres have, apparently, escaped on the left side, while the tumour still transgresses a little the middle line.

Fig. 3 shows the tumour still farther invading the right side. This section passes through the olivary body; but, unfortunately, the whole of the anterior pyramid is wanting on the left side. Yet this is not of so much consequence, for we know that those descending fibres have already been cut off above.

In Fig. 4 the tumour occupies nearly equal portions of the medulla on either side. The olive has only been indicated on one side, as the section was too thick on the other side for it to be traced. Neither can I say which is right and which left side. It will be noticed that the tumour encroaches very much on the fourth ventricle. Evidently, the two halves of the medulla have been forced apart from behind by the tumour as by a wedge, hence the displacement towards the middle line of the outer end of the olive.

Below this section the tumour must have rapidly thinned out, as in places where the olive is still well developed the tumour either remains as a very small patch near the middle line, or is entirely absent.

In mid-pons the tumour is getting smaller, but I have no sections to show its vanishing point above.

One might expect that in this case there would be atrophic

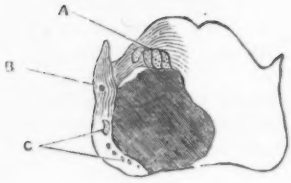

Fig. 1.

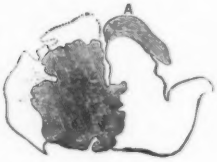

Fig. 2.

Fig. 3.

Fig. 4.

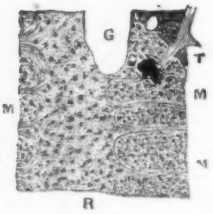

Fig. 5.

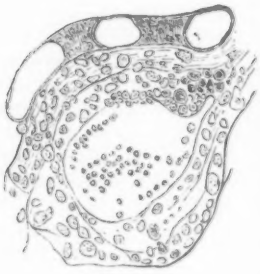

Fig. 6.

FIG. 1. Transverse section through Pons at the level of emergence of fifth nerves.

A. Bundles of descending fibres of crusta.
B. Great root of fifth nerve emerging.
C. Outlying portions of tumour.

FIG. 2. Transverse section through highest planes of medulla.

A. Lowermost fasciculi of superficial fibres of pons included in the section.

FIG. 3. Transverse section through middle of medulla. The left anterior pyramid is wanting.

FIG. 4. Transverse section through medulla below former. Part of the section is wanting behind on the right of the figure.

FIG. 5. Transverse section through edge of tumour at its lowest extremity, showing infiltration of the surrounding structures with leucocytes, magnified 37 diam.

T. Tumour.
R. Raphé with transverse bundles of fibres proceeding from it on either side.
M, M, M. Transversely cut bundles proceeding to ant. cols. of cord.
G. Gap for vessel.

FIG. 6. From edge of tumour, magnified 130 diam:, showing a giant cell imbedded in delicate stroma and smaller cells of varying size. At its apex the great cell is apparently budding off a number of small cells.

changes in the descending fibres of the medulla, but there were none present; there does not seem to have been time enough for their development.

Let us now try to trace the connections between the nervous symptoms and the structural lesions.

In the first place, as we have seen, the difficulty of swallowing was the earliest symptom of nerve lesion. This fact, taken with the other that the tumour reached the periphery of the medulla at the fourth ventricle only, seems to point to the new growth having started there.

The paralyses separate themselves into two classes—A. of cranial nerves—B. of spinal nerves.

A. The order in which the cranial nerves were affected was, (1) those presiding over deglutition, viz.—the vagi, accessories, glossopharyngeals and hypoglossals. The symptoms were, impaired deglutition, loss of speech, and tonic contractions of the sternomastoids and trapezii. (2) The left facial. The symptoms were, first, drooping of the left upper lid, which was probably spasmodic, and, subsequently, paralysis of the left side of the face. (3) The right facial. The symptoms were, occasional drooping of the right upper lid, which was spasmodic, and occasional clonic contractions of the right side of the mouth.

A nerve may have its functions interfered with in one or more of three ways. Damage may be done to (1) its nucleus or nuclei of origin; (2) the strands connecting its nucleus or its roots with the cerebral hemispheres; or (3) its roots proceeding peripherally from the nucleus.

The nuclei of the vagi, cranial accessories, and glossopharyngeals are in the floor of the fourth ventricle, or just beneath it. At the level of Figs. 3 and 4, these nuclei should exist within a small area extending outwards from the middle line; but it is here that they are quite destroyed, and also that all communication with the hemispheres is cut off. Lower down, however, where the central canal is just closed in, and the tumour is just thinning out, the lower extremity of one of the vago-accessory nuclei (the tuberculum cinereum) is quite free, and fibres of the accessory can be traced outwards and forwards from it. Close beside this latter nucleus, on its outer side, is a band of vertical fibres (the "solitary fasciculus") which ascending, according to Meynert,[1] from *crustal* fibres, probably from just above the decussation of the pyramids, crosses the raphé, and distributes its fasciculi to the rootlets of the vagus, accessory, and glossopharyngeal nerves. From the vagus and glossopharyngeal these fibres

[1] Theodor Meynert "On the Brain of Mammals." 'Stricker's Histology,' vol. ii. Syd. Soc., 1872.

would be quite cut off, but the accessory would receive contributions from it. The continuity of the *spinal* rootlets of the accessory would of course be quite free.

"Opposite to the vago-accessory nucleus, and enclosed by the transverse sections of the posterior division of the medulla oblongata, is situated the *anterior* column of origin of the *lateral mixed system* [glossopharyngeal, vagus, and accessory] in the form of an oblong nucleus lying at a distance of three millimetres from the grey substance of the central cavities. . . . This *anterior nucleus of origin* of the lateral system represents in the medulla oblongata the *processus lateralis* of the anterior cornu of the spinal column. The former allows the upper, the latter the lowermost, fasciculi of origin of the lateral system to arise from it as the accessory nerve."[1]

This nucleus is distinct from the *tuberculum cinereum*. We shall presently see the significance of this. The nuclei of the hypoglossal nerves lie in planes mostly below the level of the tumour, and, though some of their roots will pass through the tumour, many will be quite free from it. The *central* connections of the nuclei are through the raphé and the anterior pyramids of the opposite side. As the fibres of the right pyramid only are quite (if that) cut off above, the left pyramidal fibres of the nuclei will be continuous, in part at least, with the hemispheres; and as, moreover, there are commissural fibres between these two nuclei of concerted action, the central connections of both will be in part retained.

The facial nerves.—On the *left*, the tumour, at the level of Fig. 1, would have entirely obliterated the facial on that side. The area of the *right* facial nuclei and roots is throughout intact, but their central links of connection suffer partial interruption. These links are threefold. (1) From the common nucleus of the sixth and facial which, after entering the raphé as fibræ rectæ, "run obliquely downwards towards the medulla oblongata, curve round [from the posterior to the anterior part] at the lower border of the pons, and run with the pyramids into the crus cerebri"[2] of the opposite side. (2) Fibres which enter the raphé directly from the facial *root* and decussate. (3) Meynert says that the central fibres from the inferior nucleus of the facial "may connect the nucleus through the raphé with the crus cerebri." Thus all these fibres entering the *right* half of the pons from the *left* of the middle line run the risk of being invaded by the tumour growth.

Sixth nerve.—It does not seem possible for the nucleus of the *left* nerve to have escaped destruction, while the *right*

[1] Meynert, op. cit. page 506. [2] Ibid. p. 491

nerve would, like the facial, run the risk of interference with its central fibres.

Having acquainted ourselves with the foregoing anatomical facts, we are in a position to follow up some of the symptoms to their origin in the morbid processes.

First, with regard to the areas supplied by motor filaments from the eighth pair of cranial nerves. The destruction of the centres of the vagi and glossopharyngeals and, partly, of the *cranial* accessories will amply account for interference with deglutition and phonation without enquiry as to how far the hypoglossals were at fault. But what caused the loss of speech? The child had begun to speak but had left off again, and I have neither note nor recollection of his ever making the least attempt at conveying his ideas, save by the "little cracked squeal" above mentioned. Yet we have not found enough in the condition of the hypoglossals to account fully for this. I do not remember his ever being told to "put out his tongue." Could it be that the difficulty in using his larynx and lips and tongue, and his generally demoralised state, made the attempt at employment of the little speech he had acquired too irksome?

The destruction of the upper part of the anterior accessory nucleus, with infiltration of its structure by the *advancing border* of the growth lower down, might well lead to an "irritation" of its spinal portion, which would be expressed by occasional spasmodic contractions of the sterno-mastoids and trapezii.

The initial but transient drooping of the left upper lid, mentioned by the father, and the subsequent *spasmodic* drooping of the right upper lid observed by me, the occasional twitching of the right side of the mouth, and the permanent paralysis of the left side of the face, are all easily accounted for. At first the tumour only "irritated" the left facial nerve, but, as it grew, destroyed that nerve, and began the same operation on the opposite one.

As to sixth nerve. The left nerve must have been wholly paralysed, yet no squinting was observed. But this is just one of those things that might escape notice if not specially looked for, and specially looked for it was not.

B. *Paralyses of spinal nerves.*—On the left side of Fig. 2 we find that all, or nearly all, the fibres of the anterior pyramid have been destroyed—the imperfection of the specimen does not allow us to say that a very few of the anterior fibres did not escape—while in Fig. 4, some portion of the anterior pyramid remains to each side. All we can say for certain is, therefore, that only a very few fibres of the *left crusta* could have reached the *right* half of the cord. On the other hand,

although portions of the anterior pyramids do, as I say, remain in Fig. 4, all their structure to the left hand of the small vessel indicated in the figure is much infiltrated by cell-growth. Hence the left half of the cord must also have lost much of its connection with the opposite crusta. This is plain, although we cannot tell which is right and which is left in the figure.

But first of all, this question presses for answer. If the child had lost all his anterior pyramid on the left side, how was he able to move his right arm at all?

It is in man that the crusta attains its highest development, and this is always commensurate with the development of the cerebral hemispheres in animals. This is not the case with the tegmentum. Compare the figure of the transverse section of the calf's medulla given by Van Der Kolk in his treatise on the Spinal cord and Medulla,[1] with the one he gives of the medulla of Man. It will be seen that in the former the great mass of the transversely cut fibres belong to the tegmentum, while the crusta is represented by the feeble bundle of the anterior pyramid. Meynert[2] divides the tegmental fibres in the human medulla into two groups; (1) "the region of the *anterior column* forming the territory adjacent to the raphé, that is, bounded laterally . . . in the oblongata by the roots of the hypoglossus;" (2) "the region of the *lateral column* extending from this boundary as far as the ascending roots of the fifth," i.e. to the outer border. Meynert calls these fibres motor, but insists on their functional separation from those of the crusta. He points out that these latter proceed uninterruptedly from the corpus striatum to their destination in the lateral columns; whereas the former, proceeding from the optic thalami and corpora quadrigemina, become interlaced with the arcuate system of the sensory tracts, being brought into more intimate relation with them, and thus with the cerebellum, through the agency of scattered nerve corpuscles.[3] However, it is still uncertain what the function of these fibres is. May it have been that, failing its anterior pyramidal fibres, the right half of the cord was supplied with motor filaments through some of these lateral tegmental bundles of the same side? For a few would have escaped on either side of Fig. 4.

The legs were quite paralysed to voluntary movement, and the trunk nearly so. From soon after admission, onwards, their was rhythmical jactitation in both arms on any attempt at voluntary movement. A similar movement had been seen to occur in the trunk, along with that of the right arm (see p. 2). This kind of movement was exceedingly like that of

[1] New Syd. Soc., 1859. [2] Op. cit. page 467.
[3] Op. cit. pages 452, 474–78.

Multiple Sclerosis. Could it have been that the cause in each disease was the same, namely, a blocking of *some* of the motor fibres to the several muscles, but not of *all?*

As there was no coma, the effusion into the ventricles must have been gradual, and have allowed the cerebrum to accommodate itself to the pressure. Was the cause of the effusion the obstruction of the communication between the fourth ventricle and the arachnoid cavity of the cord?

The new growth was essentially infiltrating. At its periphery the proper structure of the organ can be well seen, with leucocytes lying amongst it. There was some difficulty in defining the outline of the tumour in Fig. 4; indeed, the outline given is necessarily a rough one, so much was the surrounding tissue infiltrated. In the sections from which Fig. 5 was taken, and in sections just below it, the appearances suggested, at first sight, secondary degeneration, but closer inspection showed them to be due to infiltration at the periphery of the growth. Sections a little lower were quite free from any excess of leucocytes.

The only spot where the tumour seems to be clearly separated from the surrounding normal tissue, is from the bundle A, in Fig. 1, to the emergence of the fifth nerve, i.e. along the line of contact with the superficial band of cerebellar fibres of the pons.

ARRESTED GROWTH OF THE LEFT ARM AND LEG IN AN EPILEPTIC PATIENT. LOCALISED ABSENCE OF GREY MATTER IN THE MOTOR AREA OF THE OPPOSITE CEREBRAL HEMISPHERE.

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J. F., aged 36, admitted March 10th, 1875, from Whitehaven. Is stated to have been imbecile, epileptic, and a cripple from birth. Was sent to the Asylum because he became noisy and violent, and could not be controlled at home.

On admission, he was excited and somewhat violent. He was an imbecile. Could, as far as his physical deformity allowed him, attend to his personal wants, and when in the humour could answer simple questions.

His stature generally was small (60 inches). The right side of his body was muscular and proportionately developed. The left arm was undeveloped and useless functionally. Its various parts, bones, muscles, fingers, nails, &c., were all present, but much less in size than the corresponding parts on the right side. This arm was adducted, flexed at the elbow and wrist, and the thumb rigidly flexed on the palm. The left leg was two inches shorter than the right. It was flexed at the knee, slightly so at the hip, and the heel was drawn up. All the bones were smaller and shorter than those of the opposite extremity. Some of the muscles arising from the pelvis, such as the glutei, the rectus, and the hamstrings, were fairly well developed, but below that the muscles were quite undeveloped. The foot was shorter and smaller than the right foot. He could move the limb at the hip-joint, but it was of no use to him in locomotion. He moved from place to place by hopping on his right leg, which he could do with great dexterity and endurance. Those limbs were not merely rudimentary limbs, but limbs whose growth was arrested in early life. Their bones had ossified and grown to a certain size, but the muscular tissue, from want of use, had not grown. The skin of the limbs was sensitive.

He took two or three epileptic fits in each week. The first stage of the fit was longer than usual. He uttered loud cries for twenty or thirty seconds before being convulsed. There was no convulsion of the left arm, but the muscles attached to the pelvis on the left side were more or less convulsed during a fit. He remained in the Asylum till the 9th of November, 1881, when he died of pneumonia of both lungs and epilepsy.

Post-mortem appearances.—The body was in fair condition as to fatness. The hair on the head was thick and coarse, as was the beard. The body generally, except the left arm and leg, was covered with strong thick hair. The state of the left arm and leg is described above.

Head.—Scalp thick; skull-cap thick, dense, hard; outer table much developed. The dura mater was thick and tough, its vessels engorged. On removing the dura mater a distinct depression was apparent on the upper surface of the right hemisphere in the parietal lobe. The pia mater was congested, and not so easily separable from the convolutions as usual. The brain weighed 38½ oz.; the right hemisphere 15¾ oz.; the left hemisphere 17¾ oz., and the cerebellum and pons 5 oz. The convolutions of the right hemisphere were atrophied generally, more markedly in the frontal and parietal lobes. Those of the left hemisphere were better developed, and had a more plump appearance. The depression on the surface of the right parietal lobe was in those convolutions at the upper end of the fissure of Rolando. In the marginal line it was two inches in extent from before backwards; on the outer, one and a quarter inches from above downwards, and five-eighths of an inch on the internal aspect. This depression was filled with a loose connective tissue, which was incorporated with the pia mater, and which adhered to the bottom of the depression. The posterior end of the first frontal, the upper ends of the ascending frontal and parietal convolutions, and the posterior part of the supra-marginal lobule, as far as the calloso-marginal fissure, formed the margins. At the margins of the depression the convolutions were smooth, rounded off. There was no grey matter in the bottom of the depression, the grey matter of the convolutions ending at its edges, the connective tissue being joined to the white matter of the hemisphere. There were no tracts of atrophy apparent proceeding from this depression, nor were either the ganglia, the crura, nor medullary tracts smaller on one side than the other. The left hemisphere presented no abnormality. The brain on section was firm; much congested. The cerebellum and pons were firm, congested, dark in colour.

Both lungs were in a state of red hepatisation, passing into

grey. With that exception the other organs and viscera were healthy.

Remarks.—The general impression given by the appearance of the cerebral lesion was that the brain-substance at this spot had been destroyed at birth or during very early life, probably by an injury causing effusion of blood, which became organised in time into the loose tissue which filled the depression. That the injury took place during very early life is rendered more probable by the fact that there was no trace of fracture or depression to be seen in the skull-cap.

It is of interest to note that the portion of convolutions absent or destroyed corresponds closely to the area in which Dr. Ferrier places the centres for the various complex movements of the arms and legs. These are the centres numbered by Dr. Ferrier, 2, 3, and 4.[1]

The question of connection between the ungrown limbs and the cerebral lesion naturally arises. I incline to think the cerebral lesion was the first condition produced. This caused motor paralysis of the limbs, and arrestment of growth from want of use. Probably, too, the lesion was of such a severe character as not to permit of compensatory motor action from co-ordination with the centres of the opposite hemisphere being established. A lesion of such an extent in the motor area might also produce a general nervous instability, and be the cause of the epilepsy.

All this, however, is a matter of speculation, as no accurate history of the man's early life could be obtained. The coincidence of the arrested growth of these limbs and the position of the cerebral lesion, as it more or less corroborates the results obtained by physiologists in their experimental researches into the motor functions of the cerebral surface, is, to say the least of it, somewhat remarkable.

[1] See 'Functions of the Brain,' pp. 141–2, 305.

PATHOLOGICAL CONTRIBUTIONS ON THE COURSE OF THE OPTIC NERVE FIBRES IN THE BRAIN.

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With the advance made in the study of cerebral localisation the question as to the total or partial decussation of the optic nerves in the chiasma, their further course in the tract and within the brain, and their connection with a visual centre situated in the cortex of the brain, has again come to the fore, and been made the subject of numerous observations and investigations.

The subject has been studied anatomically, by tracing the course of the fibres, and their connection with the cerebral ganglia and with the cortex; it has been studied experimentally on animals in various ways; and lastly, we have the clinical observations and the study of the lesions found after death in such cases.

Of all these different lines of investigation the clinical observations and the pathological revelations have, so far, given the most concordant results, and, while I hope shortly to consider the whole subject, in reference to these several lines of research, I wish merely in this short notice briefly to describe two cases, with post-mortem results, which have an important bearing on this subject.

The history of the first case is partially given in the 'British Medical Journal,' Aug. 7th, 1880; the patient has since then died, and I am now enabled to give the whole history of the case, with the results of the post-mortem examination, which entirely confirmed the diagnosis made *intra vitam*.

The patient was seen by me for the first time on April 3rd, 1880, at the Convalescent Hospital at Cheadle, when the following notes were taken by Mr. Stanwell, then resident Clinical Clerk.

History.—N. H., aged 40, joiner, married, with no history

of syphilis, gout, rheumatism, or alcoholic excesses, had been ill for two years, and unable to work during the last eighteen months, suffering from giddiness, headache, double vision, with numbness, weakness and trembling of the left side. He had not suffered from vomiting. The bladder and rectum had not been affected. A week before admission, he had one morning before rising three fits of distinctly epileptic character; his memory had recently become impaired, and he suffered from occasional attacks of headache, associated with febrile symptoms.

State on admission.—The patient is strongly built, sallow, fairly nourished; he has an anxious expression, and a hesitating manner. The left side of forehead is relatively smooth, while the right side is habitually wrinkled; the left palpebral fissure is slightly the larger (he can, however, close the left eye). The left angle of the mouth is rather lower than the right; the movements of the left side of the face are less extensive. The left upper limb is colder and paler, and the left forearm measures a quarter of an inch less in circumference than the right. The grip of the left hand is weaker, and the whole limb is involuntarily and frequently jerked about. These spasmodic movements affect the forearm and arm, but not the hand or the fingers; they are increased by effort or attention, and especially when any movement is attempted by the right hand (as in writing) or by any reflex excitement of the right side (such as tickling the right foot). The movements cease during sleep. The left leg is weaker than the right; it also is the seat of involuntary spasmodic movements, less in extent, but of the same character as in the left arm, and equally increased by exciting reflex contractions. The patient can walk, but in doing so drags the left leg. The patellar tendon reflex is considerably increased on the right side in both upper and lower extremities. The electric contractility is normal on both sides.

Sensation.—There is total left hemianæsthesia, exact in extent, both before and behind; the sensibility to contact, to pain, and to temperature being much diminished. The muscular sense is normal on the left side, and the patient distinguishes well different weights supported on the left hand; he can stand and walk with closed eyes. The pin-punctures bleed equally on the left as on the right side. The pupils are equal and active. There is no nystagmus or ocular palsy, but there is exact and total left hemianopsia of each field of vision. (The term "hemianopsia" is used, as Hirschberg proposed, and Mauthner and others have since adopted it, to relate to the absent field of vision, applying the term hemiopia for the insensible part of the retina—the patient

suffered, therefore, from homonymous left hemianopsia or right homonymous hemiopia). The central vision is normal; the fundi are normal; the veins of the fundus somewhat large, those in the left somewhat tortuous. Smell and taste are normal. The ticking of a watch is heard only when the watch is in contact with either ear (the deafness is, however, of old date). The urine is normal.

Diagnosis.—The symptoms of the case were so typical and well-marked, and corresponded so well with what had been seen in other cases, published, with post-mortem accounts, by Gower, Hughlings-Jackson, Hirschberg, and myself, that the case was looked upon as one of organic lesion, most likely a tumour, implicating the thalamus, the posterior part of the internal capsule, and the optic tract of the right side of the brain.

Progress.—The further progress of the case is chiefly interesting on account of the influence of the electro-magnet, the repeated application of which produced a permanent amelioration of the anæsthesia, especially of the left arm and neck, without, however, in any way interfering, even temporarily, with the hemianopsia. (For a further account of this stage in the history of the case I may refer to the account in the 'British Medical Journal' above quoted.) During the further progress of the case, there was chiefly noticed an impairment of the mental condition of the patient; he wandered about the wards at night, disturbed the rest of the patients, and was therefore removed to the Manchester Infirmary on May 26th. For a short time he remained in the same condition. Soon, however, his mental condition became worse; he became delirious and at times violent; the hemiparesis also became worse, so that he was almost completely hemiplegic; the involuntary movements ceased, the hemianopsia remained the same, and the hemianæsthesia (as far as the patient's condition would allow an opinion to be formed) remained in the same improved condition in which we found it after the repeated application of the electro-magnet. The patient gradually grew weaker, and died on Sept. 5, 1880.

The further symptoms which had been observed during his stay in the Infirmary (namely the gradually increasing weakness of the left arm and legs, the cessation of the involuntary movements and the appearance of the mental disturbances) were believed to be due to an extension of the tumour forward, implicating more of the motor tract in the internal capsule, and to softening in the neighbourhood of the tumour.

The post-mortem examination was made by Mr. A. H. Young, Pathologist to the Infirmary, to whom I am also indebted for the accompanying sketch (Fig. 1). All the organs except the

brain were firm and healthy, and here again the right hemisphere only was found affected.

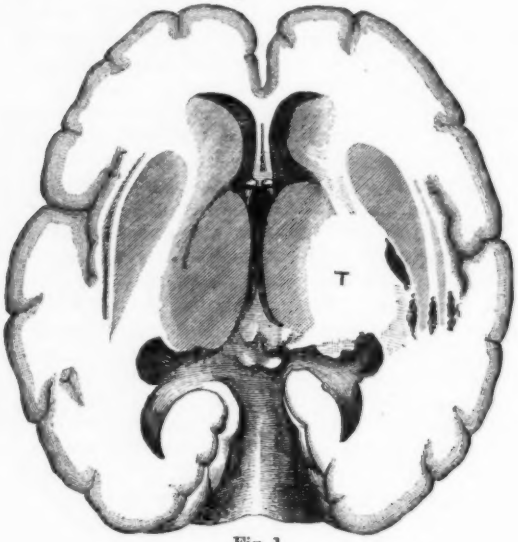

Fig. 1.

The right cerebral hemisphere appeared on superficial examination to be slightly more prominent than the opposite hemisphere, but there was no perceptible flattening of its convolutions. On transverse section of the brain there was found a tumour, diffuse in its distribution and in no way circumscribed or encapsuled, hence the determination of its exact limit was impossible. The main mass of the tumour, however, was situated in the posterior part of the internal capsule, extending from this as a centre to the optic thalamus, the lenticular nucleus, and the anterior of the corpora quadrigemina of the right side; all of which were infiltrated with new growth, and felt somewhat denser than normal. The anterior corp. quadrigem. was distinctly enlarged. The optic thalamus and lenticular nucleus were partly replaced by tumour-masses, partly simply pushed aside, so that their distal margins were more unduly separated than those of the corresponding opposite bodies (see Fig. 1). On examining the base of the brain, the right optic tract, just where it crosses the crus, and where itself becomes hidden by the gyrus hippocamp., was found flattened, and somewhat softer than the corresponding left tract.

The tumour was not, however, solid throughout. There was an irregular cavity in its interior, which was opened by the last incision made to expose the tumour, and it appears in the sketch as an oval opening to the inner side of the lenticular nucleus near the posterior end of the latter structure, and involving also the posterior extremity of the claustrum; the substance of the brain was softened and presented a loose reticular appearance.

Microscopic sections of the tumour showed it to be of a sarcomatous nature. It consisted chiefly of round cells, with a reticulum of fine fibrillæ, a few spindle cells, numerous blood-spaces, and embryonic blood-vessels.

The medulla and spinal cord, examined after hardening, showed the characteristic descending scleroses. I have recently again examined the sections of the cord, with the view of finding the sclerosed patch seen by Gowers in a case where there was anæsthesia, and which is situated anterior to the lateral pyramidal tract, but I found in this case no sclerosis on the spot indicated.

The symptoms observed during life are, as will at once be evident, satisfactorily explained by the situation of the tumour found in the brain. The tumour chiefly and primarily occupying the posterior part of the internal capsule explains the hemianæsthesia. The paresis and involuntary contractions observed during the first course of the disease are chiefly due, most likely, to an irritation of the motor fibres in the anterior part of the internal capsule, from the growth and extension of the tumour anteriorly. This state of irritation gave place to a state of paralysis, and hence we notice, during the later progress of the case, the disappearance of the involuntary movement, and the increase of the paralysis of the left side.

As for the hemianopsia, this can be accounted for in more than one way. We have noticed that the right optic tract was softened and flattened; though microscopically examined, it presented no decided changes. This would, perhaps, be enough to account for the hemianopsia; though we know, on the other hand, that cerebral nerves will often, by slow pressure, become compressed, without, however, becoming impaired in their function. The thalamus opticus, again, was found affected to a very great extent; and this, especially when considered with the second case to be described, and with some recent cases recorded,[1] might in itself be sufficient to account for the hemianopsia. Lastly, the right anterior ant. corp. quadrigem. was found enlarged and infiltrated

[1] (Pflüger, 'Bericht der Berner Augenblick,' 1818; Remy, Nothnagel, Topische Diag., &c., p. 231. Hughlings-Jackson and Gowers, Ophthalm. Hosp. Rep., vol. viii., p. 330.)

with new growth, and its connection with the optic tract is sufficiently well known. While thus the case does not teach us much as regards the course of the optic nerves in the basal ganglia, it offers an important support to the theory which advocates partial decussation of the optic nerves, and from this point of view deserves to be placed on record.

The second case I wish to put on record is one in which the lesion which produced the hemianopsia consisted of a small hæmorrhagic focus situated in the posterior portion of the thalamus opticus, without in any way affecting either the tract, the corp. geniculata, or the corp. quadrig. For the purposes of localisation, this case, therefore, is of great importance.

In describing the history of the case and post-mortem appearances I can be very brief.

Mary B., married, aged 50, was admitted into the Convalescent Hospital at Cheadle on April 2nd, having been transferred thither from the Manchester Infirmary, where she had been under the care of Dr. Simpson—suffering from Bright's disease and mitral stenosis. During her stay at the Infirmary she had some œdema and ascites, which, however, had entirely disappeared on her admission into the Convalescent Hospital. On the morning of April 16th she had an apoplectic attack, ushered in by loss of consciousness; and when this had passed away, it was found that the patient had some amount of aphasia, which was, however, but transient, and left hemiplegia. Two days after the attack I saw the patient, in conjunction with Dr. Grant, the resident Medical Officer at Cheadle, and noticed the following conditions. The patient has left hemiplegia, complete as far as the arm and leg are concerned, the face is but little affected. She complains of numbness in her left side and the sensibility seems slightly diminished on that side; her intelligence is clear, her speech but little affected. Occasionally she cannot find the exact word she wishes to express, but otherwise speech is unaltered. She complains of not being able to see well, and on examination it is found that she has left hemianopsia, the hemianopsia in each eye not reaching quite up to the point of fixation, but coming very near to it. The line limiting the hemianopsia is almost vertical, and the loss of vision within the area of hemianopsia absolute. The central vision of the patient is fairly normal, the fundus of the eye normal, the pupils are equal and react well to the light. There is no affection of any other of the cerebral nerves. The other special senses are normal.

The tendon reflexes on the paralysed side are slightly increased. There is no paralysis of either bladder or rectum. The area of cardiac dulness is increased, and over the apex a distinct systolic blowing murmur is heard, very limited in

extent; the second aortic sound is much accentuated. The urinary secretion is profuse, and contains albumen and granular casts. The temperature of the body since the attack has been above the normal, showing evening exacerbations.

I saw the patient again on the 23rd of April, and found her in almost the same condition as on the previous visit, except for the hemiplegia, which was now less marked, the patient being able to move her hand and forearm slightly. The hemianopsia had remained in the same state. The patient complained of neither headache nor giddiness.

She remained in this state till April 29, when she became somewhat suddenly drowsy, then comatose, and died the same day.

The post mortem was made on April 30 by Mr. Kaye, resident Clinical Clerk at Cheadle, who found the kidneys small and granular; the heart very much hypertrophied, especially the left ventricle; the mitral valve narrowed, but without any endocarditic deposits. The brain and its membrane showed externally normal relations, except the left lobe

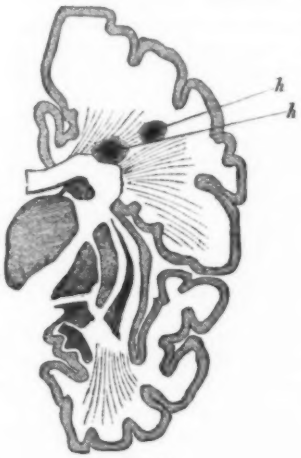

Fig. 2.

of the cerebellum, where a freshhæmorrhage was found on the under surface of the lobe, just beneath the pia mater, forming a thin layer of about two lines in thickness and an inch in circumference. On making successive sections through the brain, the following lesions were found: Two small spots of hæmorrhage, *h. h*, Fig. 2, circular in outline and of ½ inch diameter in the

centr. ovale of right hemisphere, corresponding to the ascending frontal convolution. (The accompanying drawing, Fig. 2, made by Mr. A. H. Young, shows these lesions in M. Pitre's diagram.) A little behind these two hæmorrhages, corresponding nearly to the ascending parietal convolution, was another small hæmorrhage, smaller than the two just described, but again situated entirely in the centr. ovale, encroaching neither upon the cortex nor upon the basal ganglia. On making further sections through the brain, a small circular hæmorrhage, measuring about four lines in the larger transverse diameter and three lines in the vertical direction, was seen at the posterior and upper extremity of the right thalamus opticus, the part described as the pulvinar, not, however, reaching the upper surface of the thalamus, and therefore quite within its substance. The corpora quadrigemina, the corp. genicul., and the optic tract are found perfectly intact, nor did the rest of thalamus in the immediate neighbourhood of the hæmorrhage show any changes.

The diagnosis made during life was: "hæmorrhage into the right thalamus opticus, affecting the internal capsule," and it was thought that there was only one hæmorrhagic focus.

The post-mortem examination showed that there were several spots of hæmorrhage, that the internal capsule was perfectly intact, and that the hemianæsthesia and the hemiplegia were due to separate hæmorrhages; for on comparing the symptoms during life with the lesions observed after death, I think there can be little doubt that the following is the correct view. The hemiplegia was due to the several hæmorrhages in the centrum ovale, for these are situated in that part of the white substance which contains the motor fibres radiating from the internal capsule to the motor area of the cortex (see Fig. 2). The hemianopsia was due to the hæmorrhage into the thalam. opticus. The hæmorrhage on the under surface of the left lobe of cerebellum, which was of more recent date than the other hæmorrhages, cannot well be connected with either the hemiplegia or the hemianopsia; it probably came on the last day of life, when the patient was noticed to become drowsy and comatose.

What makes this case especially interesting is the definite and well-circumscribed lesion in the thal. optic. with which the hemianopsia must be connected. *Gowers* has observed hemianopsia in many cases of cerebral hæmorrhage independently of the seat of lesion; in all these cases, however, the hemianopsia was temporary only, and soon passed away. In our case, on the other hand, the hemianopsia was permanent and, as regards its extent, constant.

Now it is admitted on all hands that fibres from the optic

tract (chiefly of its external root) pass either directly or indirectly (through the external geniculate bodies, and according to some also through the anterior corp. quadrigem.) into the thalam. opticus, and thence to that portion of the cortex of the brain which forms the visual centre. Some other fibres, however, particularly those of the internal root, and the fibres described by Stilling as passing into a ganglion situated in the peduncle of the brain, do not traverse the thal. optic. If one may therefore draw conclusions from this case, it would seem that nearly all those fibres of the optic tract which are concerned in vision pass, after having undergone semidecussation in the chiasma, whatever their connection with the corp. quadrig. and corp. genicul. may be, almost in their entirety through the thal. optic. on their way to the cortical visual centre.

Looking over the literature of the subject, I find that there are a few cases on record in which lesions of the thalamus opticus were associated with hemianopsia; in most, if not all, the lesions were more extensive, and implicated neighbouring parts.

In Pooley's case,[1] where there was right paresis, right hemianæsthesia and right hemianopsia, a tumour was found in the occipital lobe reaching to the cortex; the thalamus opticus was found softened.

Baumgarten[2] describes a case of left hemianopsia coming on suddenly in a patient affected with Bright's disease. Death ensued after several months. An old apoplectic cyst was found in the right occipital lobe and thalam. opticus.

The only other point I wish to draw attention to in our case is the condition of the pupil. This was found to react well to light, which would be quite in accordance with the view held by many that the centre for the reflex movements of the iris is situated neither in the cortex nor in the thalam. opticus, but most probably in the anterior corp. quadrigemina. I mention this, as it is important from a diagnostic point of view in distinguishing hemianopsia produced by a cortex lesion from hemianopsia produced by lesions of the optic tract, or of the ganglia situated in front of this reflex centre.

[1] Knapp's 'Arch.,' Bd. vi. p. 27.

[2] Centralb. f. med. Wissen.

NEUROMA OF THE MEDIAN NERVE REMOVED BY OPERATION.

BY WALTER RIVINGTON, F.R.C.S. ETC.

THOMAS B——, 29, labourer, was admitted into the London Hospital on December 22nd, 1873, with a tumour of the right arm. It occupied the inner aspect of the arm a little above the centre, and was about the size of a cricket-ball. It was hard, resisting, and only slightly elastic. It was very tender, and caused the most severe pain, which radiated along the limb, affecting especially the dorsal aspect of the middle and index fingers. No pain was felt in the palm. On the 28th of December the patient was placed under the influence of chloroform, and Esmarch's bandage was applied. A longitudinal incision was made over the tumour, through the skin and fasciæ, an investing capsule was opened, and I was then able to separate the tumour from its attachments, and to turn it out of the capsule without interfering with the nerve with which it was connected. It was bilobate, being crossed in the centre by a transverse constriction, and it had tapering prolongations at each end which seemed to attach it to the median nerve below, and to one of the cords of the brachial plexus above. Some of the loose outer parts of the capsule were removed; but I preferred leaving the greater portion of it to running any risk of interfering with the important nerves in relation with it, by endeavouring to dissect it away. A portion of the sheath subsequently sloughed, and came away. An attack of erysipelas of the *face* followed the operation, but it did not last more than a few days, and the wound healed kindly enough. At the beginning of February the patient complained of weakness of the muscles of the forearm, and galvanism was applied with benefit.

No cause could be assigned for the growth of the tumour. Two years previously the patient had fallen down a ship's hold, but he could not say whether he struck the arm or not. He first noticed the swellings six months afterwards, when it was as large as an egg. It began to be painful five months before his admission.

On cutting the tumour across, it was found to be of firm texture, and generally of a greyish-white colour, with some small yellow patches near the margin. These patches were thought to be portions of the growth undergoing fatty degeneration, but on microscopic examination they were seen to consist of small spindle-cells, mingled with fibrous tissue. The general mass of the tumour appeared to consist of fibrous tissue, and a smaller proportion of spindle-cells than was found in the aforesaid yellow patches. The tumour was more or less vascular, especially in the yellow portions. It would therefore be proper to describe the growth as intermediate between a fibroma and a spindle-celled sarcoma, or as a fibro-sarcoma.

The patient was seen about two years after the operation. He had been sent into the hospital from the out-patients, and had been admitted under Mr. Maunder. He caught sight of me in the ward, and, beckoning to me, introduced himself, and showed his arm. There had been no recurrence of the growth, and he had good use of his arm, which he said was free from pain and had gained in strength. He had come in for a small tumour at the upper and inner part of his right thigh.

Remarks.—The only remark which I wish to make on the case is to particularise the great facility afforded to the surgeon by Esmarch's bandage in the removal of tumours requiring careful and delicate dissection. Vessels and nerves can be exposed and traced, if necessary, with as much accuracy as in the dissecting-room, and the exact attachments and connections of a tumour can be made out with a precision which is impossible when blood is effused and smeared over the exposed structures.

Abstracts of British and Foreign Journals.

Semon on Abductor Laryngeal Paralysis.—Dr. Felix Semon (*Archives of Laryngology*, No. 3, July, 1881) discusses the fact, which has been noted by various other observers besides himself, that the abductor muscles of the larynx have a special proclivity to paralysis, whether in central or peripheral disease of the vagus, spinal accessory, or recurrent laryngeal nerves.

With the exception of the crico-thyroid, as is well known, all the laryngeal muscles, adductors as well as abductors, are supplied by the recurrent laryngeal, which gives off the individual muscular branches when close to the larynx. But that there is differentiation of the centres and fibres for the various muscles is shown by the fact—and 22 cases are quoted—that in slowly progressive lesions, whether of the centres, or peripheral nerves supplying the larynx, the posterior crico-arytenoid muscles may be paralysed alone, or at least before the others are appreciably affected. On the other hand, he has not been able to find any case of *organic* disease of the nerve-centres or trunks, in which the abductors only were paralysed, though in functional neuroses the adductors are more particularly affected.

As to the cause of the special selection of the abductors in organic disease, if of central origin, we might explain it by a differentiation of the abductor from the adductor centres, though the question would remain, why should the abductor centres be specially affected? Various hypotheses might be entertained as to this, viz. the mutual co-operation of the adductor centres giving them greater resisting power than the isolated abductors; the abductors being more automatic and less under volitional control; and lastly, the analogous fact, that in central diseases the extensors are sooner affected than the flexors.

But as the same phenomenon occurs also in peripheral disease of the nerve-trunks, we may suppose either that the disposition of the nerve fibres is concentric, and the abductor fibres possibly situated at the periphery; or that there is a specific vulnerability

of the abductor filaments; or that possibly the adductors receive an increment of nerve force from the superior laryngeal nerve.

But all these hypotheses, he argues, are more or less open to objection, and he does not think that at present a satisfactory explanation is possible. Practically, however, the facts are of great value. If the vocal cords are immovable in the phonatory position, all the possibilities should be considered which might cause affection of the entire laryngeal nerve; immobility of the cords in the position of deep inspiration, if not occasioned by mechanical impairment or myopathic affection, is probably due to a functional neurosis, or local affection of the adductor twigs.

Goltz on the Functions of the Brain. Goltz (*Verrichtungen des Grosshirns; Pflüger's Archiv für Physiologie*, Bd. xxvi. Heft 1 and 2) publishes a further series of experiments on the cerebral hemispheres of the dog, in which, instead of following his old method of destroying the cortex with a stream of water, he has established the lesions by means of a White's boring machine, the construction of which is described. Several chromolithographic plates are given of the lesions and the symptoms which were observed in the different animals experimented on.

The general conclusions he arrives at are:

1. The hypothesis of circumscribed centres for special functions in the cerebral cortex is untenable.

2. There is no area of the cortex exclusively concerned with sight, hearing, smell, taste, or touch.

3. It is impossible by any circumscribed lesion of the cortex to produce permanent paralysis of any muscle, or remove it from the influence of the will.

4. The vital manifestations which we regard as indicative of intelligence, feeling, emotion, instinct, are not dependent on functionally differentiated cortical regions.

5. Destructive lesions of the frontal regions of the hemispheres cause defects which differ in certain respects from those caused by lesions of the occipital regions. These differences depend probably on simultaneous lesion of the conducting tracts which lead to the crura.

Seguin on Cerebral Localisation.—Dr. E. C. Seguin has reported several cases of considerable interest and importance as to the effects of localised cerebral lesions. His cases and remarks are contained (1) in a paper contributed to the *Transactions of the*

American Neurological Association, vol. ii., 1877, and (2) in another paper published in the *Journal of Nervous and Mental Disease*, vol. viii., July 1881.

He gives the particulars, and describes and figures the lesions in twelve cases in all. For these, reference must be made to the original papers.

The conclusions which Dr. Seguin has arrived at from these cases are :—

1. That the motor area of the cortex and allied white substance extends anteriorly as far as the lower half of the second and first frontal gyri, and posteriorly as far as the anterior part of the intra-parietal fissure. [Three cases referred to.]

2. The region lying between the limits indicated above, the middle regions of the hemisphere, on its convexity and (to a certain extent) on its median surface, including the posterior parts of the first and second, the whole of the third, frontal gyri, the whole of the ascending frontal and ascending parietal gyri, with their terminations in the longitudinal fissure known as the paracentral lobule, with probably the upper parietal lobe,—all these cortical parts, with their associated segments or fasciculi of white matter, have strong motor functions, being in direct relation with the muscles of the face, tongue, arm and leg.

This general statement is supported by the remaining nine cases, in which destructive lesions of this area gave rise to spasm or paralysis on the opposite side of the body.

He concludes more particularly :—

a. That the lower part of the third frontal gyrus is intimately connected with the organs of speech (and the function of language). Four cases are appealed to in support of this proposition.

b. The middle parts of the ascending frontal and ascending parietal gyri are directly connected with the arm of the opposite side. Two cases are cited in illustration.

c. The upper or posterior part of the ascending frontal and ascending parietal gyri, and the paracentral lobule (also the upper parietal lobule?) are directly connected with the lower and upper extremities of the opposite side, and perhaps more closely with the leg. Three cases are referred to in support of this proposition.

Tripier on the Cephalic Souffle in the Adult.—Tripier (*Revue de Médecine*—three communications—February, March, and October 1881) describes a cephalic souffle, said generally to be only

audible in children, which he has observed in the adult in cases of anæmia, chlorosis, and also in a case of intracranial tumour and in one of hydrocephalus. It is a deep systolic sound, which is diffused over the whole cranium, but which is heard in greatest intensity in the orbital region—by placing the stethoscope on the globe of the closed eye. In his first two communications he regarded the right temporal region as the point of maximum intensity, but in his last communication he states that it is the orbital region where it is best heard.

The sound is modified by pressure on the carotid, and this is perceptible both to the patient, who is aware of a subjective sound in the ear synchronous with the bruit, and to the observer. The souffle diminishes or disappears with the cure or aggravation of the disease.

There is no relation between the cephalic and cardiac souffle heard in such cases, nor is it originated in the arteries or veins in the neck.

He is of opinion that it originates in the internal carotid at the point where it enters the cranial cavity. In a case where there was a small tumour pressing against the artery here, a cephalic souffle was heard, and there was no cardiac souffle as in anæmia and chlorosis. From communication between the carotid and cavernous sinus, aneurism of the carotid or ophthalmic artery, the cephalic souffle can be diagnosed by the absence of, characteristic ophthalmoscopic appearances in the fundus, protrusion of the globe, and other signs.

The cephalic souffle does not exist in healthy adults, and Tripier believes that it may prove of great value in the diagnosis, prognosis and treatment of the cases in which it occurs.

Baginsky on the Semicircular Canals.—Baginsky (*Du Bois-Reymond's Archiv für Physiologie*, 1881, Heft 3 and 4) describes a series of experiments on rabbits and pigeons with a view to determine the nature and causes of the disorders of equilibrium and other phenomena which have been found by Flourens and other observers to be associated with injuries of the labyrinth. He injected liquids of various kinds into the tympanic cavity of the rabbit, and cut the semicircular canals individually on one or both sides of the pigeon. The results in both animals were similar. Injections of liquid always caused nystagmus and distortion of the head, temporary, or lasting till death, according to the intensity of the irritation. He is of opinion that the phenomena are due to irritation of the brain or to actual anatomical lesion. In pigeons

in most cases there were pendulum-like movements of the head, twisting of the neck, and frequently nystagmus, coming on directly after the injury of the labyrinth, or showing themselves a few days after. The cause of these he regards to be extravasations in the brain, or inflammation excited by the lesion. The pendulum-movements of the head in pigeons, which are not observed in rabbits, he attributes to peculiarities in organisation, and in reality are of the same nature as the distortion of the head seen in the latter. As to the exact way in which the brain is affected, he holds that by injections into the tympanic cavity the pressure in the cranium is suddenly increased, and this suddenness irritates the medulla oblongata more particularly at the point where the restiform body is in closest relation with the ascending root of the trigeminus. If the pressure is very great, sudden death may be caused by the effect on the medulla oblongata. Section of the semicircular canals in pigeons diminishes the intracranial pressure. The suddenness of this acts as an irritant on the brain.

The path by which the influences are conveyed from the labyrinth to the brain differs in the rabbit and pigeon. In the former it is by the perilymphatic apparatus and aquæductus cochleæ; in the latter it is by the endolymphatic apparatus and aquæductus vestibuli.

Baginsky is of opinion that the hypothesis of any special relation of the semicircular canals to equilibration or the sense of space must be given up, as all the phenomena connected with their lesion are to be attributed to injury of the brain.

Spamer on the Semicircular Canals.—Spamer (*Pflüger's Archiv*, Bd. xxv. Heft 3 and 4) criticises Baginsky's hypothesis, and points out among other things that the disturbances of equilibrium vary with the canal injured. There is no relation between the amount of disturbance and the amount of cerebrospinal fluid which escapes when a canal is cut. The disturbances vary with the kind of injury inflicted, being more marked on transverse than longitudinal section. Apart altogether from section of the canals, irritation by different chemical solutions causes marked results; and the effect of touching the canals with a heated point cannot be explained away by any hypothesis of direct injury to the brain, such as Baginsky assumes. In his own experiments the most careful examination failed to detect any lesion of the brain, or if such lesions were accidentally produced, the cases were considered unsuccessful experiments.

Spitzka on the Cerebellum.—Spitzka (*The Functional and Morphological Relations of the Cerebellum.* Reprint from *Chicago Med. Review*, July 1881) endeavours to arrive at the functions of the cerebellum by a consideration of its relative development in different animals, and of its structural relations. The cerebellum is not, as has been frequently stated, more highly developed in the lower than in the higher animals. The contrary is the case. The cerebellar tracts, viz. anterior, middle, posterior peduncles, auditory nerve-roots and trapezium, are all centripetal. The first two, though efferent as regards the cerebellum, are afferent to the cerebrum. Hence it would seem that a direct motor innervation is entirely excluded from the functional possibilities of this organ. The restiform tract is derived from the posterior columns of the spinal cord and direct cerebellar tract, and passes to the cortex of the cerebellum, a small portion, according to Stilling, going to the nucleus dentatus. On the inner side of the restiform tract there is a bundle of fibres, which he regards as being the ideal continuation of the columns of Burdach. These go to the nucleus fastigii of the vermis. The auditory nerve-root sends fibres both to the nucleus dentatus and n. fastigii. These fibres are interrupted by the auditory nucleus, and the connections between the cerebellar ganglia and the auditory nucleus are more numerous than the fibres of the auditory nerves themselves, indicating an important connection between the internal ear and cerebellum.

The trapezium joins the restiform tract. It appears to be derived from the tegmental fibres of the medulla oblongata, and goes to the vermis and nucleus fastigii. Perhaps also the fifth and pneumogastric nerves have direct cerebellar connections. The anterior peduncles (præpedunculi) run from the nuclei dentati and decussate with each other into the nuclei tegmenti. The middle peduncles (pontibrachium) form the transverse fibres of the pons, and are continued to the cerebrum in the pes pedunculi. Besides these there is an important connection, not yet clearly made out, between the cerebellum and corpora quadrigemina, viz. the valve of Vieussens, which probably has a special relation to visual co-ordination.

From a consideration of these structural connections, Spitza is of opinion that the cerebellum, whether the lowly vermis of reptiles and marsupials, or the highly developed organ of the higher animals, constitutes a sort of deflection or meeting-point for impressions originating in the auditory and tactile end-organs. Its main object must therefore be the fusion of these sensations, or

their correlation in some way for the benefit of the cerebral hemispheres. The cerebellum is not to be considered the centre for, but "the informing depôt of the finer co-ordinations engaged in maintaining the equilibrium." The cerebellum cannot act per se, but only in connection with the higher centres.

If it is asked why the sensory tracts going to the cerebellum should not go direct to the cortex, the answer is that the position of the associating ganglion of the auditory and tactile impressions was primarily conditioned by the termination of the auditory nerve-root. "Just as the cerebellum may be considered at least in its higher form as a luxuriant hypertrophy superimposed on the auditory ganglion, so the cerebrum is known to be a supplantation of the olfactory lobe."

Féré and Demars on Ménière's Disease and its Treatment.—Féré and Demars (*Revue de Médecine*, Oct. 1881) describe the symptoms, and give the details of several cases of Ménière's disease with particular reference to the influence of quinine on them, according to the method suggested by Charcot.

When a patient with Ménière's disease is treated with sulphate of quinine in doses of from 60–80 centigrammes (10–12 grains) the cinchonic sounds in the ears are added to the subjective sounds of the disease, and the patient's symptoms are rather increased. But if the quinine is omitted for eight or ten days, the sounds and the vertigo diminish notably. If the quinine is readministered for a similar period of from eight to ten days, there is a new exasperation of the symptoms, but of less intensity than at first. At the second cessation there is a very considerable amelioration, and this may go on to complete cessation of the vertiginous attacks.

Kolnessikow on the Pathology of Hydrophobia.—Kolnessikow (*Virchow's Archiv*, Bd. lviii., Heft 3, Sep. 1881) discusses the morbid appearances found in hydrophobia, and after a resumé of the results of other observers, proceeds to give an account of the changes he has observed in 20 dogs examined by him in the three years 1878–1880. His investigations relate exclusively to the condition of the brain and nervous system—the condition of the other organs and viscera being entirely in accordance with the observations of Rudnew, Lange and others.

Macroscopically the condition observable most commonly was a hyperæmia of the pia mater of the brain and cord. In a few cases there was an œdematous condition.

The microscopical appearances of the individual cases he generalises as follows:—

The changes are found regularly and chiefly in the walls of the blood-vessels. The vessels are distended with a mass of red blood-corpuscles, and the vascular walls, as in all acute encephalitic processes, exhibit proliferation of the nuclei and infiltration of the whole thickness of wall with round cells. The round-cell infiltration was seen also along the vessels and round the nerve cells in the cerebral hemispheres, spinal cord, and medulla oblongata, in insular patches. These changes in the vascular walls and interstitial tissue vary in intensity, probably dependent on the intensity of the infection, and the relative duration of the malady. The changes are particularly manifest in the corpora striata, medulla oblongata, and spinal cord. They are not so pronounced in the hemispheres, and are more frequently circumscribed than diffuse. In all cases there were found between the layers of the vascular wall and around them a large number of amyloid or hyaloid, occasionally pigmented, colloid exudation flakes and conglomerations. These consisted partly of extravasated fluid elements and blood-corpuscles, and partly of infiltrated lymphoid elements which had traversed the vascular wall. With the change in form there was also a chemical change, as most of these elements became amyloid in character, and a few colloid and pigmented. Besides these flakes and masses, there were more rarely in certain vessels, protoplasma elements (the cells of the adventitia), which contained in their protoplasm red blood-corpuscles and finely-granular pigment.

Meyer on Diphtheritic Paralysis.—Paul Meyer (*Virchow's Archiv*, Bd. lviii. Heft 2) describes the appearances he has met with in a well-marked case of diphtheritic paralysis. He has found that the peripherical nervous system exhibits well-marked signs of parenchymatous neuritis, the characters of which he describes in detail and figures.

The alteration was best seen in the branches to the diaphragm and to the abdominal muscles (including the cremaster). Next to these came the nerves to the ilio-psoas, deltoid, triceps; then those of the glutæi and adductors of the thigh. After these came the nerves to the quadriceps, and the muscles of the forearm and leg. In general the alteration is very diffuse, and there is no special predilection for any particular group, such as flexors or extensors.

Degeneration was also found in the cutaneous nerves, but to a less extent than in the motor branches.

The cranial nerves also exhibited well-marked signs of degeneration, specially well seen in the oculo-motor, abducens, and glosso-pharyngeal.

In the large nerve-trunks the degeneration was not so pronounced as in the nerves of the face and neck. The degeneration appeared to be inversely proportional to the calibre of the nerve. Degeneration was also visible in the anterior and posterior spinal roots, more particularly in the cervical and lumbar region than in the dorsal. The anterior roots were relatively more affected than the posterior.

Degeneration was also to some extent evident in the multipolar cells of the cervical and lumbar region of the cord. The cells of the posterior cornua were also affected to some extent.

The author concludes that the diphtheritic infection attacks the nervous system in general, both spinal and peripheral, and that there is no reason to regard the one as secondary to the other.

DAVID FERRIER.

Leegard on the Degeneration of Nerves and their Electrical Reactions.—(*Arch. Klin. Med.* 1880.)—It is notorious that writers differ greatly in their accounts of nerve degeneration and regeneration. This may partly depend upon the nature of the primitive lesion. Our author employed only the ligature, which he carefully applied to the nerve without disturbing its natural relations. The thread is tied, and immediately removed, so as simply to produce a solution of continuity in the axis and myelin, the empty sheaths remaining intact. The microscopical examination of the nerves was made on specimens macerated for twenty-four hours in osmic acid, and then stained in picrocarmine and other agents. In three or four days the myelin begins to run together into cylindrical masses. The axis breaks at places; hence the loss of electrical excitability. *Secondary* to these changes appears an enlargement of the nuclei in the sheath. The myelin gradually undergoes further segmentation, and ultimately (three weeks) granular degeneration. The axis breaks up also, and every trace of it is lost between the tenth and fifteenth day.

There is an increase in the number, as well as in the size, of the nuclei. At both extremities they present masses of protoplasm which tend to form a continuous streak along the sheath. This is the first step towards regeneration. Eichhorst and Neumann thought that the products of disintegration of the medulla and axis underwent a kind of chemical alteration, and that this new sub-

stance persisted in the sheath. But it is more probable that those products are altogether absorbed. It is very likely that some fibres are completely destroyed, sheath and all, and that this is the origin of the granular débris noticed among degenerating fibres.

The process of degeneration does not follow a centrifugal direction, but attacks the whole peripheral portion of the injured nerve at once. At the point of injury itself there may be some inflammatory changes, if the operation has not been conducted with due care; otherwise there occurs nothing but an increase of the nuclei. Immediately above this point the central end of the divided medulla and axis offer much the same alterations as the peripheral end on a length of 3–5 mm. or more. The first interannular segment is altered as above described; the next three or four are shorter, irregular, with their medulla broken up. The sheath is empty at places, and displays nuclear proliferation.

As mentioned above, the first steps of the process of regeneration are made before the degenerative process is complete, and starts in the protoplasma accumulated within the sheaths in the neighbourhood of the nuclei. Fine greyish threads become differentiated in the interannular segments, and run together into a continuous fibre. The new segments appear to be more numerous than in the healthy nerve. It is possible that wholly new fibres may be reproduced, but this is not certain. The supposition that several new fibres may originate in a single original one is baseless. The regeneration is the more rapid, the more peripheral the lesion has been.

The irritability of the *nerve* undergoes the following changes. There is frequently a slight increase of it during the first day after the operation. This, however, is not constant, and is followed by a diminution which becomes more marked until the nerve does no longer react to the most powerful stimuli. This occurs, on an average, on the third day. The period of unexcitability lasts a variable time, according to the depth of the lesion; and voluntary motion, as we know, returns before any electrical reactions can be obtained, especially below the point of lesion. Applied here, the galvanic current sometimes brings about muscular contractions before the faradic. It is to be noticed, also, that opening contractions are more difficult to obtain than in the normal state. The author confirms the few observations previously recorded, where an inversion of the polar effects on the nerve is noted; he has found that in certain cases A.C.C. > K.C.C., and K.O.C. > A.O.C. In these, he says, every experimental error has been carefully

eliminated, but he cannot give any explanation of the occurrence. The indirect stimulation was always characterised by a rapid, healthy contraction.

With reference to *muscle*, its loss of reaction to faradism is usually complete within four days. Vulpian has rightly insisted that this is not strictly true of muscles directly excited (not percutaneously), for here fibrillary contractions persist for a long time. Section of the antagonists may be necessary to demonstrate weak contractility. To galvanis u muscles at first react less freely, soon after the injury to the nerve, but this applies less to the strength of the current to be used than to the amplitude of the contractions; in other words, it is the maximal, not the minimal, test which displays the alteration. At the same time A.C.C. approximates K.C.C. The author has not seen the same regularity in the development of the qualitative changes during the period of increased excitability, as is usually described; he has never seen A.C.C. > K.C.C. Both O.C. disappear at first; but when they return A.O.C. is always > K.O.C. They tend to overtake the C.C. during the period of increased excitability, and disappear again afterwards, sometimes permanently. During the same period the closure and opening tetanus appears only after the closure and opening contraction have been obtained. A singular fact is that the author has not been able to obtain signs of the increased mechanical excitability of muscles so generally found in the human subject, nor of Erb's "middle form" of the degenerative reactions. The course of the electrical phenomena connected with the processes of nerve and muscle degeneration may be divided into four stages, of which a tabular view (p. 565) is appended.

[The reader interested in the question will find an account of the similar process as observed in man in the reporter's "Introduction to Medical Electricity," Chapter III.; or better still in Erb's vol. on the Peripheral nerves, in Ziemssen's 'Cyclopedia.']

Langenbuch on Nerve Stretching in Ataxy and other Spinal Diseases. (*Berlin Klin. Woch.* 1881, 24-27.)—The author begins by expressing the view that locomotor ataxy is not primarily an affection of the posterior tracts, but has a peripheral origin in the sensory nerves. In all the cases in which he operated, the nerves showed, to the naked eye, signs of alteration; sometimes swelling and reddish discoloration, sometimes dryness, atrophy, and yellowish or violet discoloration. He thinks that the large number of cases of tabes, as well as the great importance

——	First Stage.	Second Stage.	Third Stage.	Fourth Stage.
Nerve.				
Motility	Absent	Absent	Returning	Good.
Reaction to galv. and far.	Increase at first, then disappears.	Absent	Returning	Further improvement. Normal formula restored.
Muscle.				
Farado-contractility .	Disappears; only fibrillary contraction.	Fibrillary only . . .	Reappears at some period .	Rises, but not to normal.
Galvano-contractility .	Diminution of C.C. Disappearance or inconstancy of O.C. Tetanus diminishes.	Increase of all C. to maximum of hyperexcitability. O.C. constant. Tetanus evident, but appears last of formula.	At first increase, then decrease of C.C. below normal. O.C. decrease and disappear. Tetanus diminishes.	Diminished irritability. C.C. recover. O.C. absent or inconstant. Eventually rise of irritability and return of normal formula. Irritability may remain permanently deficient.
Character of contracting	Rapid. Maximal often very weak.	Slow, vigorous . . .	Strength falls off. To indirect stim. rapid, to direct slow. Gradual improvement of latter.	Strength restored; sluggishness disappears.
Mechanical irritability	Diminished	Exalted (?)	Exalted (?); later diminished	Usually diminished.

of cold in the causation of disease, is rather in favour of the peripheral origin. Another consideration is the fact that in the early stage the morbid changes in the cord are limited to the part of the posterior tracts which Flechsig has shown to be the direct continuation of the peripheral nerves. The pathological process invades Goll's columns at a later period: these we know are not in direct connection with the peripheral fibres. Further extension occurs later, by contiguity, into the neighbouring lateral tracts.

According to Langenbuch, then, we have to do in ataxy with an ascending inflammatory alteration in the nutrition of nerves, roots, and columns. How is it then that the overgrowth of connective tissue (sclerosis) is proportionally much larger in the cord than in the nerves? He thinks that the answer to this question lies in the fact that the morphological character of the connective tissue is different in the two cases. The result of connective overgrowth is much more important in the cord where the nerve elements are thickly crowded together among the thin meshes of the tissue. The author is, besides, far from saying that the morbid process always invades the cord. This is well illustrated in the temporary ataxies after acute disorders. With reference to the lightning pains, their peripheral origin seems to be shown by the happy results of compression or friction along the course of nerves.

The action of nerve-stretching is twofold: it stimulates the conductivity of partially disabled nerve-fibres, and promotes resorption of morbid products. It seems to influence the whole nervous system; the pulse and respiration-rate is quickened.

The first symptom to give way is the sensation of chilliness. After a few hours, sensibility returns in the limbs, and may even reach hyperæsthesia. After a certain diminution, muscular power increases, and the ataxic symptoms disappear. During the first few days peripheral pain is present, perhaps as the result of the operation; but within the second week the patient usually becomes entirely free from pain. The symptoms connected with the rectum and genito-urinary organs are also usually much relieved. A curious phenomenon sometimes observed is profuse perspiration after the operation. The general vigour, physical and mental, of the patient is increased, but the gait remains abnormal for a certain time. The knee-phenomenon remained absent in the twenty-two cases in which the author operated.

He has performed nerve-stretching in various other cases of spinal disease—in one (lateral sclerosis) the result was very favour-

able. Two cases of general chronic pemphigus and obstinate senile pruritus were cured by the stretching of one sciatic nerve.

On the retardation of Sensory Impulses in Locomotor Ataxy. Fischer (*Berlin Klin. Woch.* 1881, 33) examined five cases of tabes with a view of determining the relative rate of sensations of touch and pain. For such experiments it is advantageous to deal with patients intelligent enough to give an accurate description of their feelings. The retardation for impressions of pain may be considerable. He found it once to extend to fifteen seconds, but states that sensations of touch may be delayed longer than those of pain. The delay may vary during the experiment; it may become less on repeating the stimulus; or, contrariwise, the stimulus may cease to be perceived at all. Retardation of the other kinds of sensations (temperature, pressure, &c.) was not clearly demonstrated. The author does not offer any theory in explanation of the retardation.

Unilateral Perspiration and Hemiatrophy, with their relation to the Nervous System. Takács (*Ctrlblatt. f. Nervhlkd.* 1881, 13) has observed a case in which the patient (a man, aged 32, left-handed) was seized with pains, weakness and stiffness in the right extremities. At the same time he ceased to perspire on the right side of the body. Pilocarpin injections produced some sweating on that side, especially on the back of the hand, where perspiration appeared more abundant than on the left hand. The whole right side of the body was, when patient came under observation, viz. two years after the beginning of the disease, much thinner than the left. There was distinct atrophy of all the right hand-muscles, which showed deficient reaction to electricity.

The author remarks that most cases of a similar nature hitherto described have been referred to hyper- or hypokinetic conditions of the sympathetic. He thinks, however, that the combination of pupillary vasomotor and trophic symptoms has its origin in the cord itself. The cilio-spinal centre accounts for the pupillary symptoms. And, according to the late researches of Adamkiewicz, perspiration is governed by the anterior grey matter of the cord. The suspension of perspiration does not depend upon mere vaso-construction, but upon paresis of special nerve action, and pilocarpin is a stimulant of such an action.

Cherchevsky on the Laryngeal Crises of Locomotor Ataxia (*Revue de Médecine*, July 1881). The author describes eighteen

cases of tabes, in which laryngeal crises occurred. He classifies the latter in three groups, according to the intensity of the symptoms:

1. In the milder form there occur fits of coughing very like those of whooping-cough, consisting in a series of rapid short expiratory efforts, followed by a deep whistling inspiration. The duration varies from a few to ninety seconds. The expectoration is very scanty.

2. In the second group the cough is much more violent, and the external manifestations (congestion of the face and eyes) much more marked. The crowing inspiration louder and more difficult. Other symptoms, such as headache, nausea, vomiting, &c., accompany the fit. The duration is longer; five to ten minutes.

3. In the severest form there is complete apnœa, with symptoms of asphyxia, unconsciousness, epileptiform convulsions. There may be local prodromal symptoms before the fit. Its duration may extend to half an hour, or even several hours.

The laryngoscope reveals no local change; and the time and number of fits varies much in different cases. Post-mortem appearances in two cases pointed to atrophy of the vagus and accessorius roots, and of the posterior pyramids, besides the usual tabetic alterations in the cord.

In the majority of instances the laryngeal crises appeared along with the early symptoms of tabes; and occasionally formed the only prominent symptom for a considerable time.

A. DE WATTEVILLE.

A Microcephale of Simian character.—Professor Tamburini showed to a Medical Congress at Reggio Emilia a microcephalic boy, named Battista, now twelve years of age. He was a foundling. When three and a half years old he had been described by Professor Lombroso (see my report on some cases of microcephaly and cretinism in the *Edinburgh Medical Journal*, August, 1875, and my book on *Idiocy*, p. 87). Dr. Tamburini's paper is a reprint from the *Archivio Italiano per le Malatie Nervose*, Fasc. I., Anno 18, 1881.

Lombroso described Battista as having a tender and delicate skin, covered with fine hair on the forehead, limbs, cheek and neck. The head was small and peaked, with a retreating forehead, and the external orbital process protuberant. The face was prominent; the incisors distant from one another, and the canines twice as big as usual. The palate was longer, and the genital organs more developed than usual. The hallux more distant and longer than the other toes.

His height was 79 centimetres; the weight of the body 7 kilogrammes. The circumference of the head was 360 millimetres; the longitudinal curve 230 millimetres; the transverse 190; the longitudinal diameter was 128; the transverse 100. The cephalic index was 78°, and the facial angle 72°.

The most striking characteristic of this child was his fondness for leaping, and great restlessness. He generally leapt with the back bent and the hands in front of him. He did not sit, but crouched upon the ground. He smelt his food before carrying it to his mouth.

In 1877 he was brought to the asylum of Voghera, owing to his habit of tearing and destroying things and killing animals. Dr. Tamburini observes that his appearance and movements no longer answered to the picture given by Lombroso taken six years before. The smallness of the head and its disproportion to the body were very striking. The light hair diffused over the face had disappeared. His attitude and movements have still something of the Simian.

On making a number of measurements and comparing them with those of Lombroso six years before, the following differences were found on the body and head. The circumference of the head stood to the stature:—

In 1871, as 45 to 100;
1877, as 36 to 100;
1880, as 32 to 100.

The circumference of the head in 1877 was 385; in 1878, 395; in 1879, 400; in 1880 it was 418. The head was oxycephalic, and there was a marked want of symmetry in bulging behind on the right side and in front on the left. Battista was very restless, and his attention was difficult to fix; he knew the names of many things, and even used phrases; but his ideas went no further than the objects around him, and he could not be taught to read or write. He was fond of music, had little attachment to particular persons, and was subject to fits of furious passion. The genital organs were more developed than that of an ordinary boy of the same age, and his sexual desires seemed to correspond.

The Optic Tracts and the Visual Centres.—Tartuferi has in the Pathological Laboratory of Turin conducted a series of experimental researches on the optic tract and the visual centres in the cerebral ganglia and cortex cerebri. From a note in the *Italia*

Medica (No. 5, 1880) it appears that he has arrived at the following conclusions:—

1. Where the corpus geniculatum externum appears to be smaller it is often dependent upon the sclerosis of the optic tract which extends over it.

2. It is not true, as up to this time has been stated by anatomists, that all the fibres of the corpus geniculatum externum belong to the optic tract. It only forms a small part of its medullary cortex, and passes over it without entering into any intimate connection.

3. The corpus geniculatum externum cannot be considered as belonging to the mesocephalic centres of the apparatus of vision. Assuredly, at least, it has not the important significance which some anatomists would attribute to it.

4. The inferior commissure of Gudden is not, as its discoverer imagines, a simple commissure between the two optic thalami and the two corpora geniculata interna.

5. The greater part of the inferior commissure of Gudden, which is in connection with the two corpora geniculata, and especially with the external one, probably goes to compose the inferior portion of the superficial white and grey layer.

6. There are no fibres of the optic tract on the surface of the optic thalamus.

7. The thalamus is not a visual centre.

8. What is called the inner root of the optic tract going to the thalamus (innere Sehhügel-wurzel of Meynert, or tiefere Wurzelbundel of Studa) does not belong to the optic tract.

9. The fibres which compose the so-called root of the corpus geniculatum internum (Huguenin) does not belong to the tract, but to the inferior commissure.

It results from these four last conclusions that of the five roots of the optic nerve recently admitted (Huguenin, 1879) there is only one real root which passes over the corpus geniculatum externum without entering into it, and goes to the anterior tubercle of the corpora quadrigemina.

10. The diminution of the punctus occipitalis is due to atrophy, and not, as Gudden says, to a sinking, owing to the diminution of the anterior tubercles of the corpora quadrigemina which lie below it.

11. The point of the occipital lobe in the rabbit constitutes the cortical centre for the apparatus of vision. This has been confirmed by experiments upon monkeys, cats and dogs, as well as by pathological observations on man.

The effect of Metals on the Sphygmograph.—Professor Orsi, in the University of Pavia (*Italia Medica*, No. 8, 1880), studied two cases of hemianæsthesia which were modified and transferred by the application of different metals. He determined to try what effect the application of metals would have upon the sphygmograph, and came to the following conclusions.

a. That the application of lead to the skin during the first two minutes only sensibly lowers by a half, and in the end by a third, the ascending line.

b. That by substituting zinc for lead the line soon rises beyond the normal limit. If the application of zinc lasts for a while, there is a notable change in the height of the line occurring along with changes in sensibility and muscular force. The tracings obtained under the action of zinc are unequal in their character. In the ascending line they are of different heights and more or less sloping.

c. That the application of the magnet and electric current at a distance causes inequality and abnormality of the sphygmographic tracings, much in the same way as the hypodermic injection of from one to two centigrammes pilocarpin, which appears to have a stimulating action on the heart. WILLIAM W. IRELAND.

Rumpf on the Action of Lymph on the Nerve Centres. (*Pflüger's Arch.* xxvi., *p.* 415.)—Rumpf maintains the correctness of his observation that the axis-cylinder of nerve is dissolved by lymph, after passing through a preliminary stage of swelling. The swelling of nerve stumps under the influence of lymph is at first of the nerve proper, and only later consists in an inflammatory hypertrophy of the connective tissue. The author was led to examine the analogous event as regards fibres and cells of the brain and cord. The results on frogs are very striking:—the skull having been opened, a portion of brain isolated, and the wound reclosed, Rumpf found forty-eight hours later that nothing remained of the isolated portion of brain but a small quantity of connective tissue. The spinal cord having been laid bare, transected above and below, and isolated from all nerves, Rumpf found at the end of twenty-four hours that all the nerve elements have began to swell, this being still more marked at the end of forty-eight hours, after which the cord became amorphous and rich in nuclei, and (five to six days) disappeared, leaving nothing but a little connective tissue. This absorption does not ensue upon section of the cord without section of its nerves, nor, when the cord

and intervertebral nerves having been cut, the cauda equina is left intact. Nor is it produced if in addition all sensory roots of the cauda are divided; whereas if its motor roots are alone divided absorption follows, thought not so rapidly or completely. He obtained similar results with hardly any interference with the circulation. These observations indicate a centripetal trophic influence, partly via sensory channels, but chiefly via motor channels, and confirm the view of Kühne and of the author (1860) that from end-plate to centre there exists a constant excitatory and trophic influence.

The author also experimented on the brain of pigeons, and found, as in the case of frogs, swelling; absorption was, however, usually complicated by suppuration.

The author concludes that, to the maintenance of centres, their union with efferent channels is necessary, and that failing this union the organ degenerates and is absorbed by the action of the lymph; also that this union alone is sufficient to preserve the otherwise isolated cord, and that even partial union suffices by vicarious action to maintain the nutrition of the whole organ. This retrograde influence is the antithesis of tonus, and Rumpf therefore terms it "retrotonus." Thus, as laid down by Sigmund Mayer, the nerve fibre, with its two end-organs, central and peripheral, constitutes a trophic as well as functional unit.

A. Waller.

END OF VOL. IV.

LONDON: PRINTED BY WILLIAM CLOWES AND SONS, LIMITED, STAMFORD STREET AND CHARING CROSS.